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# GENERAL SURGICAL

# PATHOLOGY AND THERAPEUTICS,

## In Fifty-one Lectures.

*A TEXT-BOOK FOR STUDENTS AND PHYSICIANS.*

BY

DR. THEODOR BILLROTH,
PROFESSOR OF SURGERY IN VIENNA.

WITH ADDITIONS BY

DR. ALEXANDER VON WINIWARTER,
PROFESSOR OF SURGERY IN LÜTTICH.

*TRANSLATED FROM THE FOURTH GERMAN EDITION, WITH THE SPECIAL PERMISSION OF THE AUTHOR, AND REVISED FROM THE TENTH EDITION, BY*

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# TRANSLATOR'S PREFACE.

As will be seen by the author's preface, Prof. Billroth, with the ninth edition, turns over his work to a successor chosen by himself.

While we cannot help regretting his leave-taking from active literary effort, we must admire the strong good sense of a man in being able to decide on leaving a work while in full, vigorous bloom; while the leaves of his book are almost as fresh as when first published. Prof. Winiwarter, who is the successor, is perhaps best known, in this country at least, by his contribution to the knowledge of malignant sarcoma. The additions to the book have been quite numerous; and they, as well as additions by the translator, are enclosed in brackets [ ].

In the present English edition, the articles which were in an appendix in the last edition, as well as various new matter, have been incorporated in the text or added on the ends of the appropriate chapters. It will be noticed that two German editions have appeared since Prof. Billroth's valedictory, so that his successor has had an opportunity of going very thoroughly over the work, and making all the changes he thought advisable. It is believed that the present edition represents the existing state of knowledge in pathology.

CHARLES E. HACKLEY, M. D.

NEW YORK, *November*, 1882.

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# PREFACE TO THE NINTH GERMAN EDITION.

When my publishers recently asked me to prepare a new edition of this book, after much reflection I concluded that I should not be able to do so with as much pleasure as formerly. The practice of my profession, social duties, and my work as teacher, have so absorbed my time during the past ten years that I have not followed the advance of medical science as closely as one should who was to present the results of recent workers to a new generation of students. And, trying as it was, I concluded not to attempt the work of revision myself. To my great pleasure, Prof. Alexander von Winiwarter undertook the task, and has so successfully accomplished it that the book will again for a time serve as a guide to students and practitioners in their studies of general surgical pathology and therapeutics.

This task was the more trying because this book was so peculiarly my own work. Prof. Winiwarter has piously retained my model; but I wish that in future editions he may conform more and more to his own views, and those of the present and coming surgical generations; this is the only plan that will ensure its prolonged life.

With this preface I bid farewell, as a surgical writer, to readers who have so long cheered me by their sympathy and encouragement. I may say with pride that success has crowned my attempts to excite pleasure and effort in many young men studying the noble art and science of surgery.

Many thanks to all who have aided me in these attempts.

TH. BILLROTH.

Vienna, *January*, 1880.

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# SURGICAL PATHOLOGY AND THERAPEUTICS.

## LECTURE I.

### INTRODUCTION.

Relation of Surgery to Internal Medicine.—Necessity of the Practising Physician being acquainted with both.—Historical Remarks.—Nature of the Study of Surgery in the German High-schools.

GENTLEMEN: The study of surgery, which you begin with this lecture, is now, in most countries, justly regarded as a necessity for the practising physician. We consider it a happy advance that the division of surgery from medicine no longer exists, as it did formerly. The difference between internal medicine and surgery is in fact only apparent; the distinction is artificial, founded though it be on history, and on the large and increasing literature of general medicine. In the course of this work your attention will often be called to the frequency with which surgery must consider the general state of the body, to the analogy between the diseases of the external and internal parts, and to the fact that the whole difference depends on our seeing before us the changes of tissue that occur in surgical diseases, while we have to determine the affections of internal organs from the symptoms. The action of the local disturbances on the body at large must be understood by the surgeon, as well as by any one who pays especial attention to diseases of the internal organs. *In short, the surgeon can only judge safely and correctly of the state of his patient when he is at the same time a physician.* Moreover, the physician who proposes refusing to treat surgical patients, and to attend solely to the treatment of internal diseases, must have some surgical knowledge, or he will make the grossest blunders. Apart from the fact that the country physician does not always have a colleague at hand to whom he can turn over his surgical patients, the life of the patient often depends on the correct and instantaneous recognition of a surgical disease.

When blood spouts forcibly from a wound, or a foreign body has entered the windpipe, and the patient is threatened with suffocation, then surgical aid is required, and quickly too, or the patient dies. In other cases, also, the physician ignorant of surgery may do much harm by not recognizing the importance of a case; he may allow a disease to become incurable, and by his deficient knowledge cause unspeakable injury, in a case which might have been relieved by early surgical treatment. Hence it is inexcusable for a physician obstinately to stick to the idea of only practising internal medicine; still more inexcusable is it, in this idea, to neglect the study of surgery: "I will not operate, because in ordinary practice there is so little operating to be done, and I am not at all suited for an operator!" As if surgery consisted only in operations. I hope to give you a better idea of this branch of medicine than is conveyed by the above remark, which unfortunately is too popular.

From the fact that surgery has to deal chiefly with patent diseases, it certainly has an easier position in regard to anatomical diagnosis; but do not regard this advantage too highly. Besides the fact that surgical diseases also often lie deeply hidden, more is demanded from a surgical diagnosis and prognosis, and even in the treatment, than from the therapeutic action of internal medicine. I do not deny that in many respects internal medicine may hold a higher rank, just on account of the difficulties it has (and often so brilliantly overcomes) in localizing and recognizing disease. Very fine operation of the mind is often necessary to come to a proper conclusion, from the combination of symptoms, and the results of the examination. Physicians may point with pride to the anatomical diagnosis of diseases of the heart and lungs, where the careful student succeeds in giving as accurate a description of the changes in the diseased organ as if he had it right under his eyes. How wonderful it is to gain an accurate knowledge of the morbid state of hidden organs, such as the kidneys, liver, spleen, intestines, brain, and spinal marrow, by the examination of a patient and the combination of symptoms! What a triumph to diagnose diseases of organs of which we do not know even the physiological function, as of the supra-renal capsules! This is some compensation for the fact that, in internal medicine, we must more frequently acknowledge the impotence of our treatment than is the case in surgery, although, from the advances in anatomical diagnosis, we have become more certain of what we can do, and of what we cannot.

The irritation of the finer, cultivated portions of the mind in internal medicine is, however, richly balanced by the greater certainty and clearness of diagnosis and treatment in surgery, so that the two branches of medical science are exactly on a par. And it must not be forgotten that the anatomical diagnosis—I mean the recognition of the pathological changes in the diseased organ—is only one *means* to the end, which is the cure of the disease. *The true problems for the physician are to find out the causes of the morbid process, to prognosticate the course, conduct it to a favorable termination, or control it, and these are equally difficult in internal and external medicine.* Only one thing more is required of the practical surgeon: this is, the art of operating. This, like every art, has its knack; the facility of operating secondarily depends on accurate knowledge of anatomy, on practice, and on personal aptitude. This aptitude may also be cultivated by persevering practice. Just remember how Demosthenes succeeded in acquiring fluency in speaking.

This knack, which is certainly necessary, has long separated surgery from medicine in the strict sense; we may historically follow this separation as it constantly became more practically felt, till in this century it was finally recognized as impractical and was abolished. The word "chirurgery" at once expresses that originally it was regarded as entirely manual, for it comes from χείρ and ἔργον, which literally mean "hand-work," or, in the pleonasm of the middle ages, "hand-work of chirurgery."

Little as it comes within the scope of this work to give a complete sketch of the history of surgery, it still seems to me important and interesting to give you a short sketch of the external and internal development of our science, which will explain to you some of the various regulations affecting the so-called "medical staff" still existing in different states. A fuller history of surgery can only be of use to you hereafter, when you shall have acquired some knowledge of the value or worthlessness of certain systems, methods, and operations. Then, in the historical development of the science, especially as regards operative surgery, you will find the key for some surprising and for some isolated experience, also for much that is incomplete. Many things that may be necessary for the comprehension of the subjects, I shall relate to you when speaking of the different diseases; now, I shall only present a few prominent points in the development of surgery and of its present position.

Among the people in former times, the art of healing was intimately associated with religious education. The Hindoos, Arabs, and Egyptians, as well as the Greeks, considered the art of healing as a manifestation made by the gods to the priests, and then spread by tradition. Philologists were not agreed as to the age of the Sanscrit writings discovered not long since; formerly their origin was placed at 1000–1400 B. C., now it is considered certain that they were written in the first century of the Christian era. The Agur-Veda (" Book of the Art of Life ") is the most important Sanscrit work for medicine; it is the production of *Susrutas*. It very probably originated in the time of the Roman Emperor Augustus. The art of healing was regarded as a whole, as is indicated by the following: "It is only the combination of medicine and surgery that makes the complete physician. The physician lacking knowledge of one of these branches is like a bird with only one wing." At that time surgery was without doubt by far the more advanced part of the medical art. A large number of operations and instruments are spoken of; still, it is truly said "the best of all instruments is the hand;" the treatment of wounds given is simple and proper. Most surgical injuries were already known.

Among the Greeks all medical knowledge at first centred in *Æsculapius*, a son of Apollo, and a scholar of the Centaur Chiron. Many temples were built to Æsculapius, and the art of healing was handed down by tradition through the priests of these temples; the number of these temples induced various schools of Æsculapides, and, although every one entering the temple as a priest had to take an oath, which has been handed down to our own times (although of late its genuineness appears rather doubtful), that he would only teach the art of healing to his successors, still, as appears from various circumstances, even at that time there were other physicians besides the priests. From one part of the oath, even, it is evident that then as now there were physicians who, as specialists, confined themselves to certain operations; for it says: "Furthermore, I will never cut for stone, but will leave this operation to men of that occupation." Of the different varieties of physicians we know nothing more accurate till the time of *Hippocrates;* he was one of the last of the Asklepiades. He was born 460 B. C., on the island of Cos; lived partly in Athens, partly in Thessalian towns, and died 377 B. C. at Larissa. We might expect that medicine would be considered scientifically at this time, when the names of Pythagoras, Plato, and Aristotle, were shining in Grecian science; and in fact the works of Hippocrates, many of which are still preserved, arouse our astonishment. The clear classical description, the arrangement of the whole material, the high regard for the healing art, the sharp critical observations, that appear in the works of Hippocrates, and compel our admiration and respect for ancient Greece on this branch also, clearly show that it is not a case of blind belief in traditional medical dogmas, but that there was already a scientific and elaborately perfected medicine. In the Hippocratic schools the art of healing formed one whole; medicine and surgery were united, but there were various classes of medical practitioners; besides the Asklepiades there were other educated physicians, as well as more mechanically instructed medical assistants, gymnasts, quacks, and workers of miracles. The physicians took scholars to train in the art of healing; and, according to some remarks of Xenophon, there were also special army physicians; especially in the Persian wars, they, together with the soothsayers and flute-players, had their places near the royal tent. It may be readily understood that, at a time when so much was thought of corporeal beauty, as was the case among the Greeks, external injuries would claim special attention. Hence, among physicians of the Hippocratic era, fractures and sprains were particularly studied; still, some severe operations are treated of, as also numbers of instruments and other apparatuses. They seem to have been very backward regarding amputations; probably the Greeks preferred dying to prolonging life after they were mutilated The limb was only removed when it was actually dead, gangrenous.

The teachings of Hippocrates could not at first be carried any further, for lack of knowledge of anatomy and physiology. It is true there was a faint effort made in this direction in the scientific schools of Alexandria, which flourished for some centuries under the Ptolemies, and by means of which, after the wars of Alexander the Great, the Grecian spirit was spread, at least temporarily, over part of the Orient; but the Alexandrian physicians soon lost themselves in philosophical systems, and only advanced the science of healing a little by a few anatomical discoveries. In this school the art of healing was at first divided into three separate parts—dietetics, internal medicine, and surgery. Along with Grecian culture, their knowledge of medicine was also brought to Rome. The first Roman physicians were Grecian slaves; the freedmen among them were allowed to erect baths; here, first, barbers and bathers became our rivals and colleagues, and for a long time they injured the respectability of the profession in Rome. Gradually the philosophically-minded took possession of the writings of Hippocrates and the Alexandrians, and themselves practised medicine, without, however, adding to it much that was new. The great lack of original scientific production is shown in the encyclopedial revision of the most varied scientific works. The most celebrated work of this nature is the "*De Artibus*" of *Aulus Cornelius Celsus* (from 25–30 B. C. to 45–50 A. D., in the time of the Emperors Tiberius and Claudius). Eight books of this, "*De Medicina*," have come down to our time; from these we know the state of medicine and surgery at that time. Valuable as are these relics from the Roman ages, they are only, as we have said, a compendium, such as is often published at the present day. It has even been denied that *Celsus* was a practising physician, but this is improbable; from his writings we must, at all events, credit *Celsus* with using his own judgment. The seventh and eighth books, which treat on surgery, could not have been written so clearly by any one who had no practical knowledge of his subject. Hence we see that, since the time of *Hippocrates* and the Alexandria school, surgery, especially the operative part, had made no great progress. *Celsus* speaks of plastic operations, of hernia, and gives a method of amputation which is still occasionally employed. One part, from the seventh book, where he speaks of the qualifications of the perfect surgeon, is quite celebrated, as it is characteristic of the spirit which reigns in the book; I give it to you: "The surgeon should be young, or at least little advanced in age, with a hand nimble, firm, and never trembling; equally dexterous with both hands; vision, sharp and distinct; bold, unmerciful, so that, as he wishes to cure his patient, he may not be moved by his cries to hasten too much, or to cut less than is necessary. In the same way let him do every thing as if he were not affected by the cries of the patient."

*Claudius Galenus* (131–201 A. D.) must be regarded as a phenomenon among the Roman physicians; eighty-three undoubtedly genuine medical writings of his have come down to us. *Galen* returned again to the Hippocratic belief, that observation must form the foundation of the art of healing, and he advanced anatomy greatly; he made dissections chiefly of asses, rarely of human beings. *Galen's* anatomy, as well as the entire philosophical system into which he brought medicine, and which seemed to him even more important than observation itself, has stood firm over a thousand years. He occupies a very prominent position in the history of medicine. He did little for surgery in particular; indeed, he practised it little, for in his time there were special surgeons, either gymnasts, bathers, or barbers, and so unfortunately surgery was handed down by tradition as a mechanical art, while internal medicine was, and long remained, in the hands of philosophic physicians; the latter knew and commented freely on the surgical writings of *Hippocrates*, the *Alexandrians*, and *Celsus*, still they paid little attention to surgical practice. As we are only giving a faint sketch, we might here skip several centuries, or even a thousand years, during which surgery made scarcely any progress, indeed retrograded occasionally. The Byzantine era of the empire was particularly unfavorable to the advance of science, there was only a short flickering up of the Alexandria school. Even the most celebrated physicians of the later Roman times, *Antyllus* (in the third century), *Oribasius* (326–403 A. D.), *Alexander of Tralles* (525–605 A. D.), *Paulus of Ægina* (660), did relatively little for surgery. Some advance had been made in the position and scholarly attainments of physicians; under Nero there was a gymnasium; under Hadrian an athenæum, scientific institutions where medicine also was taught; under Trajan, there was a special medical school. Military medical service was attended to among the Romans, and there were special court physicians, "archiatri palatini," with the title of "perfectissime," "eques," or "comes archiatrorum," just as, among the Germans, "Hofrathe," "Geheimrathe," "Leibarzte," etc. That, as a result of the fall of science in the Byzantine reign, the art of healing did not totally degenerate, is due to the Arabians. The wonderful elevation that this people attained under Mohammed, after the year 608, aided in preserving science. The Hippocratic knowledge of medicine, with the later additions to it, passed to the Arabians through the Alexandrian school, and its branches in the Orient, the schools of the Nestorians; they cherished it till their power was demolished by Charles Martel, and returned it to Europe by way of Spain, though somewhat changed in form. *Rhazes* (850–932), *Avicenna* (980–1037), *Albucasis* († 1106), and *Avenzoar* († 1162), are the most celebrated, and for surgery the most important, of the Arabian physicians whose writings have been preserved; the writings of the latter are the most important for surgery. Operative surgery suffered greatly from the dread the Arabians had of blood, which was partly due to the laws of the Koran; it caused the employment of the actual cautery to an extent that we can hardly comprehend. The distinction of surgical diseases and the certainty of diagnosis had decidedly increased. Scientific institutions were much cultivated by the Arabians; the most celebrated was the school of Cordova; there were also hospitals in many places. The study of medicine was no longer chiefly private, but most of the students had to complete their studies at some scientific institution. This also had its effect on the nations of the West. Besides Spain, Italy was the chief place where the sciences were cultivated. In southern Italy there was a very celebrated medical school at *Salerno;* it was probably founded in 802 by Charles the Great, and was at its zenith in the twelfth century; according to the most recent ideas, this was not an ecclesiastical school, but all the pupils were of the laity. There were also female pupils, who were of a literary turn; the best known among these was *Trotula.* Original observations were not made there, or at least to a very slight extent, but the writings of the ancients were adhered to. This school is also interesting from the fact that it is the first corporation that we find having the right to bestow the titles "doctor" and "magister."

Emperors and kings gradually took more interest in science, and founded universities; thus universities were founded in Naples in 1224, in Pavia and Padua in 1250, in Paris in 1205, in Salamanca in 1243, in Prague in 1348, and they were invested with the right of conferring academical honors. Philosophy was the science to which most attention was paid, and for a long time Medicine preserved her philosophical robe in the universities; in some cases they adhered to Galen's system, in others to the Arabian or to new medico-philosophical systems, and registered all their observations under these heads. This was the great obstacle to the progress of the natural sciences, a mental slavery, from which even men of intellect could not free themselves. The anatomy of *Mondino de Luzzi* (1314) differs very little from that of Galen, in spite of the fact that the author bases it on dissections he made of some human bodies. In surgery there were no actual advances; *Lanfranchi* (†1300), *Guido of Cauliaco* (beginning of the fourteenth century), *Branca* (middle of the fifteenth century), are a few of the noteworthy surgeons of those times.

Before passing to the flourishing state of the natural sciences and of medicine in the sixteenth century, we must review briefly the composition of the medical profession in the times of which we have been speaking, as this is important for the history. First, there were philosophically educated physicians either lay or monk, who had learned medicine in the universities or other schools; i. e., they had studied the old writings on anatomy, surgery, and special medicine; they practised, but paid little attention to surgery. Another seat of learning was in the cloisters; the Benedictines especially paid a great deal of attention to medicine and also practised surgery, although the superiors disliked to see this, and occasionally special dispensation had to be obtained for an operation. The regular practising physicians were sometimes located, sometimes travelling. The former were usually educated at scientific schools and received permission to practise on certain conditions. In 1229, the emperor Frederick II. published a law that these physicians should study logic (that is, philosophy and philology) three years, then medicine and surgery five years, and then practise for some time under an older physician; before receiving permission to practise independently, or, as an examiner lately said, of physicians who had just received their degree, "till they were let loose on the public." Besides these located physicians, of whom a great part were "doctor" or "magister," there were many "travelling doctors," a sort of "travelling student" who went through the market-towns in a wagon with a merry Andrew, and practised solely for money. This genus of the so-called charlatans, which played an important part in the poetry of the middle ages, and is still gleefully greeted on the stage by the public, carried on a rascally trade in the middle ages; they were as infamous as pipers, jugglers, or hangmen; even now these travelling scholars are not all dead; although, in the nineteenth century, they do not ply their trade in the market-place, but in the drawing-rooms as workers of miracles, especially as cancer-doctors, herb-doctors, somnambulists, etc. Let us now inquire the relation, of those who practised surgery, to the above company. This branch of medicine was occasionally resorted to by almost all of the above; still there were special surgeons, who united into guilds and formed honorable societies; they received their practical knowledge first from a master, under whom they studied, and subsequently from books and scientific institutions. Surgical practice was chiefly confined to these persons, who were mostly located, but sometimes travelled about as "hernia doctors," "operators for stone," "oculists," etc. We shall become acquainted with some excellent men among these old masters of our art. Besides the above, surgery was also practised by the "bathers," and later by "barbers" also, as it was among the Romans, and they were permitted by law to attend to "minor surgery," e. g., they could cup, bleed, treat fractures, sprains, etc. It will be readily understood that some strife would arise about the various and sometimes indefinite privileges of these different grades of physicians, especially in large cities, where all classes of them were collected. This was particularly the case in Paris. The surgical society there, the "Collége de St. Côme," claimed the same privileges as members of the medical faculty; they were particularly desirous for the Baccalaureate and Licentiate. The "Society of Barbers and Bathers," again, wished to practise any part of surgery, just like the members of the Collége de St. Côme. To gall the surgeons, the members of the faculty supported the claims of the barbers, and, in spite of mutual temporary compromises, the strife continued; indeed, we may say that it still continues, where there are pure surgeons (surgeons of the first class and barbers) and pure physicians. It is only since about 1850 that the distinction was done away with in almost all the German states, and neither chirurgi puri nor medici puri were made, but only physicians who practised medicine, surgery, and obstetrics.

To finish the question of external rank, we may notice that in England alone there is still a tolerably well-marked dividing-line between surgeons and physicians, especially in the cities, while in the country "general practitioners" attend to both medical and surgical cases, and have an apothecary-shop even at the same time.

In Germany, Switzerland, and France, circumstances often cause a physician to have more surgical than medical practice; but the medical staff legally consists of physicians and assistants or barber-surgeons, who, after examination, are licensed to cup, bleed, etc. This arrangement has finally gone into effect in the army also, where the so-called company surgeon, with the rank of sergeant, formerly had a miserable time under the battalion and regimental physicians.

In again taking up the thread of the historical development of surgery, as we enter the period of "Renaissance" in the sixteenth century, we must first think of the great change which then took place in almost all sciences and arts, on account of the Reformation, the discovery of printing, and the awakening spirit of criticism. Observation of Nature began to reassume its proper position and gradually but slowly to free itself from the fetters of the schools; investigation after truth again assumed its claims to being the only true way to knowledge—the Hippocratic .spirit was again awakened. It was chiefly the new investigations, we might almost say the rediscovery, of anatomy and the subsequent restless progress of this branch, that levelled the road. *Vésal* (1513–1564), *Falopia* (1523–1562), and *Eustachio* (†1579), were the founders of our present anatomy; their names, like those of many others, are known to you from the appellations of certain parts of the body. The celebrated *Bombastus Theophrastus Paracelsus* (1493–1554) was among the first to criticise the prevailing Galenical and Arabic systems, and to claim observation as the chief source of medical knowledge. Finally, when *William Harvey* (1578–1658) discovered the circulation of the blood, and *Aselli* (1581–1626) discovered the lymphatic vessels, the old anatomy and physiology were obliged to give place to modern science, which thence gradually progressed to our times. Even then it was a long time before practical medicine escaped in the same way from philosophic thraldom. System was founded on system, and the theory of medicine constantly varied to correspond to the prevailing philosophy. We may claim that it was not till pathological anatomy made its great advances in the present century that practical medicine acquired the firm anatomico-physiological foundation on which the whole structure now moves, and which forms a strong protection against all philosophical medical systems. Even this anatomical direction, however, may be pushed too far and too exclusively. We shall speak of this hereafter

Now we will turn our attention to the scientific development of surgery from the sixteenth century to our times.

It is an interesting feature of that time that the advance of practical surgery depended more on the surgical societies than on the learned professors of the universities. German surgeons had to seek their knowledge mostly in foreign universities, but part of it they worked out for themselves independently: *Heinrich von Pfolsprundt*, a German friar (born the beginning of the fifteenth century), *Hieronymus Brunschwig* (born 1430), *Hans von Gersdorf* (about 1520), and *Felix Würtz* (†1576), surgeons at Basel, are first among these. We have writings of all of them; *Felix Würtz* seems to me the most original of them; he had a sharp, critical mind. *Fabry von Hilden*

(1560–1634), of Berne, and *Gottfried Purman*, of Halberstad and Breslau (about 1679), were men of great acquirements; their writings show a high appreciation for their science, they fully recognized the value and imperative necessity of exact anatomical knowledge, and by their writings and private instruction imparted it to their scholars as much as possible.

Among the French surgeons of the sixteenth and seventeenth centuries, *Ambroise Paré* (1517–1590) is most prominent; originally only a barber, from his great services, he was made a member of the Society of St. Côme; he was very active as an army surgeon, was often called from home on consultations, and at last resided in Paris. *Paré* advanced surgery by what was for those times a very sharp criticism of treatment, especially of the enormous use of problematical remedies; some of his treatises, e. g., on the treatment of gun-shot wounds, are perfectly classical; he rendered himself immortal by the introduction of ligature for bleeding vessels after amputation. *Paré*, as the reformer of surgery, may be placed by the side of *Vésal*, as reformer of anatomy.

The works of the above individuals, besides some others more or less gifted, held their place into the seventeenth century, and it is only in the eighteenth that we find any important advances. The strife between the members of the faculty and those of the Collége de St. Côme still continued in Paris; the great celebrity of the latter had more effect than the professors of surgery. This was finally practically acknowledged in 1731 by the foundation of an "Academy of Surgery," which was in all respects an analogue of the medical faculty. This institution soon advanced to such a point that it ruled the surgery of Europe almost a century; nor was this an isolated cause; it formed part of the general French influence, of that universal mental dominion which the "grande nation" cannot even yet forget when German science has forever eclipsed French influence, after the conflicts of 1813–'14. The men who then stood at the head of the movement in surgical science were *Jean Louis Petit* (1674–1768), *Pierre Jos. Desault* (1744–1795), *Pierre François Percy* (1754–1825), and many others in France; in Italy, *Scarpa* (1748–1832) was the most active. Even in the seventeenth century, surgery was highly developed in England, and in the eighteenth century it attained great eminence under *Percival Pott* (1713–1768), *William* and *John Hunter* (1728–1793), *Benjamin Bell* (1749–1806), *William Cheselden* (1688–1752), *Alexander Monro* (1696–1767), and others. Among these was *John Hunter*, that great genius, as celebrated for anatomy as surgery; his work on inflammation and wounds still forms the basis of many of our present views.

In comparison with these, the names of the German surgeons of the eighteenth century are insignificant; most of them brought their knowledge from Paris, and added little that was original: *Lorenz Heister* (1683–1758), *John Ulrich Bilguer* (1720–1796), and *Chr. Ant. Theden* (1719–1797), are relatively the most important. German surgery only obtained greater eminence with the commencement of the present century. *Carl Caspar von Siebold* (1736–1807), and *August Gottlob Richter* (1742–1812), were distinguished men; the former served as professor of surgery in Würzburg, the latter in Göttingen; some of *Richter's* writings are valuable even now, especially his little book on rupture.

On the threshold of our century you see professors of surgery again in the foreground, where they subsequently maintained their position, because they actually practised surgery. A predecessor of old *Richter*, as professor of surgery at Göttingen, the celebrated *Albert Haller* (1708–1777), at once physiologist and poet, one of the last encyclopædists, says, "Etsi chirurgiae cathedra per septemdecim annos mihi concredita fuit, etsi in cadaveribus dificilimas administrationes chirurgicas frequenter ostendi, non tamen unquam vivum hominem incidere sustinui, nimis ne nocerem veritus." To us this seems scarcely credible, so great is the change wrought by a hundred years. Even at the commencement of this century the French surgeons remained at the helm; *Boyer* (1757–1833), *Delpech* (1776–1832), and particularly *Dupuytren* (1777–1835), and *Jean Dominique Larrey* (1776–1842), were almost undisputed authorities in their line. Besides them there arose in England the unimpeachable authority, Sir *Astley Cooper* (1768–1841). *Larrey*, the constant companion of Napoleon I., left a large number of works; you will hereafter read his memoirs with great interest. *Dupuytren* was chiefly celebrated for his excellent clinical lectures. *Cooper's* monographs and lectures will fill you with astonishment. Translations of the writings of the above French and English surgeons first aroused German surgery; but soon the subject was gone into most profoundly by original workers. The men who induced the German revolution in surgery were, among others, *Vincenz von Kern*, of Vienna (1760–1829), *John Nep. Rust*, of Berlin (1775–1840), *Philipp von Walther*, of Munich (1782–1849), *Carl Ferd. von Graefe*, of Berlin (1787–1840), *Conr. Joh. Martin Langenbeck*, of Göttingen (1776–1850), *Joh. Friedrich Dieffenbach* (1795–1847), *Cajetan von Textor* (1782–1860), of Würzburg.

The nearer we approach the middle of our century, the more the rugged bounds of nationality disappear from the domains of surgery. With increased means of communication, all advances in science spread with breathless haste to all parts of the civilized world. Numberless writings, national and international medical congresses, and personal intercourse, have brought radical changes to the surgeons as well as to others. A generation of surgeons, upon whose works the profession looks with honor, appears to be now dying out; I mean men such as *Stanley* (1791–1862), *Lawrence* (1783–1867), and *Brodie* (1783–1862), in England; *Roux* (1780–1854), *Bonnet* (1809–1858), *Leroy* (1798–1861), *Malgaigne* (1806–1865), *Civiale* (†1867), *Jobert* (1799–1868), and *Velpeau* (1795–1867), in France; *Seutin* (1793–1862), in Belgium; *Valentine Mott* (1785–1865), in America; *Wutzer* (1789–1863), *Schuh* (1804–1865), and others, in Germany. From our own generation also we have some losses to mourn, especially the irreparable death of the gifted, indefatigable investigator *O. Weber* (1827–1867); of the excellent *Follin* (–1867), one of the most solid of modern French surgeons; of *Middeldorpf* (1824–1868), the celebrated inventor of galvano-caustic operations. Among the living we might name many on whose shoulders rests the growing generation of German surgeons, but they do not yet belong to history. But there is one point I must not leave unmentioned, that is, the introduction of pain-quelling remedies into surgery. The nineteenth century may be proud of the discovery of the practical use of sulphuric ether and chloroform as anæsthetics in all sorts of operations. In 1846 came from Boston the first news that *Morton* the dentist, at the suggestion of his friend *Dr. Jackson*, had, in extracting teeth, employed inhalations of sulphuric ether, pushed to complete anæsthesia, with perfect success. In 1859, *Simpson*, professor of obstetrics in Edinburgh, instead of ether, introduced in surgical practice chloroform, which acts still better, which, after various trials with other similar substances, still preserves its reputation. Thanks! in the name of suffering humanity, a thousand thanks to these men!

In continuation of my previous remarks regarding German surgery, I will simply add that at present it stands at least equal to that of other nations, and is perhaps even superior to that of France at the present time. To perfect ourselves in the science of surgery, we no longer need to visit Paris. But, of course, it is nevertheless desirable for every physician to enlarge his experience and observation by visiting foreign lands. In the scientific as well as in the practical part of surgery, and of medicine generally, England is now more advanced than any other country. In America also great advances have been made in practical surgery. From the time of *Hunter* to the present day, English surgery has about it something noble. Surgery owes its great revolution in the nineteenth century to its attempt to unite all medical knowledge in itself; the surgeon who succeeds in this, and also masters the entire mechanical side of the art, may feel that he has attained the highest ideal in medicine.

Before entering on our subject, I will add a few remarks about the study of surgery as it is, or is said to be, pursued in our high-schools.

In the four years' course of medical study which is customary in German universities, I would advise you not to begin surgery before the fifth semestre. You often desire to escape the preliminary studies and plunge at once into the practical. It is true, this is somewhat less the case since courses on anatomy, microscopy, physiology, chemistry, etc., have been started in the high-schools, where you have some practice; nevertheless, there is still too much haste to enter the clin ics. It is true, it is one way of gaining experience from the very start; you consider it more interesting than bothering yourselves at first with things whose connection with practice you do not exactly understand. But you forget that a certain school of observation must be gone through with, to enable us to make actually useful what we know. If any one just released from school should at once enter the hospital as a student, he would be in the same position as a child entering the world to collect knowledge. Of what use are the experiences of the child for his subsequent life among men? How late it is before we see the true use of the most common observations of daily life! Hence, to wade through the entire development of medicine in this empirical manner would be a long, tedious labor, and only a very gifted, industrious man would learn any thing in this way. After having made numerous errors, we must not place too great a value on "experience" and "observation," if by these terms we mean no more than the laity do. It is an art, a talent, a science, to observe critically, and from our observations to draw correct conclusions for our "experience;" this is the strong point of the empiric; the laity know experience and observation in the vulgar, not in the scientific sense, and they value the so-called experience of an old shepherd as high as, sometimes higher than, that of a physician; unfortunately, the public are sometimes right on this point. But enough! when a physician or any one else displays his experience and observation before you, look sharply to see whether he has any brains.

In making these remarks against pure empiricism, we do not by any means intend to say that you must be theoretically acquainted with all medicine before studying it practically, but you should bring a certain knowledge of the fundamental principles of natural science with you into the clinic. It is absolutely necessary to have a general idea of what you are to expect; and you must know something of the tools before seeing them used, or taking them in your hands. In other words, you should know the outlines of general pathology and therapeutics, as well as of materia medica, before going to the bedside of the patient. General surgery is only one part of general pathology, hence you should study the latter before entering the surgical clinic. First, you should gain a clear understanding of normal histology, at least of its general parts; pathological anatomy and histology should come with general surgery, about the fifth semestre.

General surgery, the subject of the present lectures, is a part of general pathology, as we have already stated; but it is nearer to practice than the latter. It comprises the study of wounds, inflammations, and tumors of the external parts of the body, or of those parts that may be handled from without. Special or topographical surgery occupies itself with the surgical diseases of different parts of the body, so that the most different tissues and organs are to be considered according to their location; for instance, while we here treat only of wounds, of their mode of recovery and treatment in general, special surgery treats of wounds of the head, breast, and abdomen, paying special attention to the participation of the skin, bones, and viscera. Were it possible to pursue the study of surgery for several years in a large hospital, and could careful clinical consideration of individual cases be carried on continuously with the regular studies, it would probably be unnecessary to treat of special surgery in separate systematic lectures. But, since there are many surgical diseases that perhaps may not occur for years even in a large hospital, but which should be known to the surgeon, the lectures on special surgery are by no means superfluous, if they are short and to the point.

During my student days I occasionally heard the remark: "Why should I go to listen to special surgery and pathology? I can read them more conveniently in my room." This may be all true, but unfortunately it is rarely done, unless in the final semestres, when examination is approaching. This reasoning is false in another respect also: the *viva vox* of the teacher, as old *Langenbeck*, in Göttingen, used to say (and he had a *viva vox* in the best sense of the word), the winged word of the teacher is, or should be, more exciting and effective than what is read, and the accompanying demonstrations of diagrams, preparations, experiments, etc., should render the lectures on practical surgery and medicine particularly valuable for you. I attach great value to demonstration in medical instruction, for I know by experience that this kind of teaching is most exciting and permanent.

Besides these two sets of lectures on general and special surgery you have to practise operations on the cadaver; this you may postpone to the last semestres. I always like students to take their course in operations in the sixth or seventh semestres, along with their special surgery, so that I may give them the opportunity of occasionally operating, or even of amputating, under my direction. It gives courage in practice, if one has during student-life performed operations on the living subject. When you have followed the lectures on general surgery, you may enter the surgical clinic, and there, in the seventh and eighth semestres, openly give an account of your knowledge in special cases, and accustom yourselves to collecting your ideas rapidly, learn to distinguish the important from the unimportant, and to learn generally in what practice really consists. You will thus learn the points where your knowledge is deficient, and may perfect yourselves by persevering study. When you have thus completed the legal time of your studies, passed your examination, and have increased your medical knowledge by a few months or a year in various large hospitals at home or abroad, you will be in condition to appreciate the surgical cases turning up in practice. But, if you wish to devote special attention to surgery and operating, you are still far from the goal: then you must become accustomed to operating on the cadaver, enter a surgical ward as assistant for a year or two, untiringly study monographs on surgical subjects, perseveringly write out cases, etc.—in short, follow out the practical school from the lowest step. You must be fully acquainted with hospital service, even with the duties of the nurses; in short, you should know practically even the most minute things appertaining to the care of patients, and should even perform the duties yourselves occasionally, so that you may be fully master of the entire medical service intrusted to you.

You see there is much to do and to learn: with patience and perseverance you will accomplish it all; but these virtues are necessary to the study of medicine.

"Student" comes from "to study;" hence you must study faithfully; the teacher indicates to you what he considers the most important; he may stimulate you in various directions; what he gives you as positive may, it is true, be carried home in black and white, but, to cause this positive knowledge to live in you and become your mental property, you must depend on your own mental efforts, which form the true "study."

When you conduct yourself as a passive receptacle, you may, it is true, acquire the name of a very "learned person," but, if you do not awake your knowledge into life, you will never become a good "practising physician." Let what you see enter your mind fully, warm you up, and so occupy your attention that you must think of it frequently, then the true pleasure and appreciation of this mental labor will fill you. *Goethe*, in a letter to *Schiller*, aptly says: "Pleasure, comfort, and interest in the affairs of life, are the only realities all else is vanity and disappointment."

# CHAPTER I.

## *SIMPLE INCISED WOUNDS OF THE SOFT PARTS.*

## LECTURE II.

Mode of Origin and Appearance of these Wounds.—Various Forms of Incised Wounds.—Appearance during and immediately after their Occurrence.—Pain, Bleeding.—Varieties of Hæmorrhage; Arterial, Venous.—Entrance of Air through Wounded Veins.—Parenchymatous Hæmorrhage.—Hæmorrhagic Diathesis.—Hæmorrhage from the Pharynx and Rectum.—Constitutional Effects of Severe Hæmorrhage.

The proper treatment of wounds is to be regarded as the most important requirement for the surgeon, not only on account of the frequency of this variety of injury, but because we so often intentionally make them in operating, even when operating for something that is not itself dangerous to life. Hence we are answerable for the healing of the wound, to as great an extent as it is possible by experience to judge of the danger of an injury. Let us commence with incised wounds.

Injuries caused by sharp knives, scissors, sabres, cleavers, hatchets, etc., represent pure incised wounds. Such wounds are usually recognizable by the regular sharp borders, where we see the smooth-cut surface of the unchanged tissue; should the instruments be blunt, by very rapid motion they may still cause quite a smooth incised wound, while by slowly entering the tissue they would give the edges of the wound a ragged appearance; occasionally, the variety of the injury does not become evident till the wound is healing, for wounds made with sharp instruments heal more readily and quickly (for reasons to be given hereafter) than those caused by the slow entrance of dull knives, scissors, etc.

Rarely a perfectly blunt body makes a wound exactly like an incised one. This may occur from the skin being torn through by force applied through a blunt object, at a point where it lies over the bone. Thus you will not unfrequently see scalp-wounds resembling incised wounds, although they may have been due to a blow from a blunt body, or from striking the head against a stone, beam, etc.; similar smooth wounds of the skin also occur on the hand, especially on the volar surface. Sharp angles of bone may so divide the skin from within that it will look as if cut through, as, for instance, when one falls on the crest of the tibia, and it divides the skin from within outward. As may be readily understood, sharp splinters of bone perforating the skin may also make wounds with smooth surfaces. Lastly, the opening of exit of a bullet-wound, i. e., of the canal which represents the passage of a bullet, may sometimes be a sharp slit.

The knowledge of these points is important, for a judge may ask you if a wound has been caused by this or that instrument, in this or that manner, points which may greatly affect the bearings in a criminal suit.

Hitherto we have only considered wounds made with a blow or stroke. But, by repeated cuts on a wound, the edges may acquire a hacked appearance, and thus the requirements for recovery may be very much changed. For the present, we leave such wounds out of consideration; their mode of recovery and treatment is just the same as that in contused wounds, unless they can be artificially converted into simple incised wounds by paring off the jagged edges. The various directions in which the cutting instrument enters the body generally makes little difference, unless the direction be so oblique that some of the soft parts are detached in the form of a more or less thick flap. In these *flap*-wounds, the width of the bridge, uniting the half-separated portion with the body, is important, because on this depends the question as to whether circulation of blood can continue in this flap, or if it has ceased, and the detached portion is to be regarded as dead. Flap-wounds are chiefly due to cuts, but may also arise from tearing; they are very frequent in the head, where part of the scalp is torn off by a hard blow. In other cases a portion of the soft parts may be entirely cut out; then we have a *wound with loss of substance.*

By *penetrating wounds* we mean those by which one of the three great cavities of the body or a joint is opened; they are most frequently due to stabs or gun-shot injuries, and may be complicated by wounds of the viscera or bones. By the general terms *longitudinal* and *diagonal* wounds we of course mean those corresponding to the long or diagonal axes of the trunk, head, or extremities. Diagonal or longitudinal wounds of the muscles, tendons, vessels, or nerves, are of course those dividing these parts longitudinally or diagonally. The symptoms in the person wounded, induced more or less directly by the wound, are, first, *pain;* then, *bleeding* and *gaping* of the wound.

As all the tissues, not excepting the epithelial and epidermoid, are supplied with sensory nerves, injury at once causes *pain.*

This pain varies greatly with the nerve-supply of the wounded part, and with the sensitiveness of the patient to pain. The most sensitive parts are the fingers, lips, tongue, nipples, external genitals, and about the anus. Doubtless, each of you knows from experience the character of the pain from a wound, as of the finger. The division of the skin is the most painful part; injury of the muscles and tendons is far less so; injury of the bone is always very painful, as you may find from any one that has recovered from a fracture; it has also been handed down to us from the times when amputations were made without chloroform, that sawing the bone was the most painful part of the operation. The mucous membrane of the intestines, on being irritated in various ways, shows very little sensitiveness, as has been occasionally observed on man and beast; the vaginal portion of the uterus also is almost insensitive to mechanical and chemical irritation; occasionally, it may be touched with the hot iron, as is done in treating certain diseases of this part, without its being felt by the patient. It appears that the nerves requiring a specific irritation, as the nerves of special sense, are accompanied by few if any sensory fibres. The relation of the sensory nerves of touch to the sentient nerves in the skin cannot be regarded as decided, or whether there be any decided difference between them. In the nose and tongue, we have sensory and sentient nerves close together, so that in both parts, besides the specific sense peculiar to the organ, pain may also be perceived. The white substance of the brain, although containing many nerves, is without feeling, as may be seen in many severe injuries of the head. The division of nerve-trunks is the severest of all injuries. Some of you may remember the pain from rupture of a dental nerve on extraction of a tooth. Severing of thick nerve-trunks must cause overpowering pains. Sensitiveness to pain appears peculiar to individuals. But you must not confound this with various exhibitions of pain, and with the psychical power of suppressing, or at least limiting, this exhibition; the latter depends on the strength of will, as well as on the temperament, of the individual. Vivacious persons display their pain, as well as their other feelings, more than phlegmatic persons. Most persons maintain that crying, as well as the instinctive powerful tension of all the muscles, especially of the masseters, gritting the teeth, etc., renders the pain more endurable. Personally, I have not been able to verify this statement, and I think it must be a mistake of the patients. Strong will in the patient may do much to suppress the show of pain. I well remember a woman in the Göttingen clinic, when I was a student, who, without chloroform, had the whole upper jaw removed for a malignant tumor, and, during this difficult and painful operation, she did not once cry out, although several branches of the trifacial nerve were divided. Women generally stand suffering better and more patiently than men. But the necessary exercise of psychical strength not unfrequently causes subsequent fainting, or excessive physical and psychical relaxation, of longer or shorter duration. You will certainly meet persons who, without any exercise of will, show so little pain from severe injury that we can only believe that they really feel pain less acutely than others; I have observed this most in flabby sailors, in whom all the sequelæ of the injury are also generally very insignificant.

The quicker the wound is made, and the sharper the knife, the less the pain; hence, in large and small operations, it has always seemed, and very correctly too, for the advantage of the patient, that the incisions should be made with certainty and rapidity, particularly in dividing the skin.

The feeling in the wound, immediately after its reception, is a peculiar burning. It can scarcely be termed any thing but the feeling of being wounded; there are a number of provincialisms for it—in Northern Germany, for instance, they say "the wound smarts." Only when a nerve is compressed by something in the wound, twisted or irritated in some way, there are severe neuralgic pains immediately after the injury; if these do not soon cease spontaneously, or after examination of the wound and removal of the local cause, if possible, they should be arrested by the exhibition of some internal remedy; otherwise, they will induce and keep up a state of excitement in the patient that may increase to maniacal delirium.

*To avoid the pain in operations*, we now always use anæsthetics; this subject will be treated of in the course on operations. Recently ether has come more into use on account of the number of deaths from chloroform. I now use a composition of 3 parts chloroform, 1 sulphuric ether, and 1 absolute alcohol, which seems less dangerous than chloroform alone. In England, for some years, *Spencer Wells*, among others, has used and recommended bichloride of methyline, claiming that it acts as quickly as, and is less dangerous than, chloroform. *Local anæsthetics*, which have for their object temporary blunting of the pain in the part to be operated on, by application of a mixture of ice and saltpetre, or salt, have been again abandoned, or rather they have never been generally received. Recently these attempts have again acquired a general interest, as it seemed that a suitable method of local anæsthesia had at last been found. An English physician, *Richardson*, constructed a small apparatus, by which a stream of pure ether [or, better, rhigoline] spray is for a time blown against one spot in the skin, and such cold is here induced that all sensation is lost. After procuring some of this ether (hydramyläther) from England, I was satisfied of its perfect action. In a few seconds the skin becomes chalky white, and absolutely without sensation; but the effect hardly extends through a moderately thick cutis; and, if the ether be still blown against the cut surface, the frozen tissues cannot be distinguished from each other, and the knife, being coated with ice, will no longer cut. Hence, even in this more perfect form, local anæsthesia can only be used advantageously in a few minor operations. My former dread, that healing of the wound would be essentially interfered with by this freezing of the part, has been shown by experience to be groundless. For quelling the pain, and as a hypnotic, immediately after extensive injuries or operations, there is nothing better than a quarter of a grain of muriate or acetate of morphia; this quiets the patient, and, even if it does not make him sleep, he feels less pain from his wound. Quite recently hydrate of chloral (ʒ ss–ʒ j, in half a glass of water) has been used; its narcotic action was discovered by *Liebreich*, 1869. Its effect is essentially hypnotic, but very uncertain; it cannot supplant chloroform, but is a decided acquisition to our materia medica. Locally, for the relief of pain, we employ cold in the shape of cold compresses, or bladders filled with ice, applied to the wound. We shall refer to this under the treatment of wounds. Lastly, we may give hypodermic injections. If, with a very fine syringe, furnished with a lance-shaped, sharp canula, which may be thrust readily through the skin, we inject a solution of $\frac{1}{8}$–$\frac{1}{4}$ of a grain of acetate or muriate of morphia, this remedy will exercise its narcotic effect at first locally on the nerves it comes in contact with, and then on the brain, as the solution is absorbed and enters the blood. Of late, this mode of employing morphia has been exceedingly popular; immediately after an operation, or severe injury, such an injection is given, and the pain is at once arrested.

In a pure incised or punctured wound, *hæmorrhage* is another immediate symptom; its extent depends on the number, size, and variety of the divided vessels. At present we shall only speak of hæmorrhage from tissues previously normal, and distinguish capillary, parenchymatous, arterial, and venous hæmorrhages, which must be considered separately.

As is well known, the different parts of the body vary greatly in vascularity, especially in the number and size of the *capillaries*. In spots of equal size the skin has fewer and smaller capillaries than most mucous membranes; it also has more elastic tissue and muscles, by which (as we may feel and see in the cold and so-called goose-flesh) the vessels are more readily compressed than they are in the mucous membranes, which are poor in elastic and muscular tissue; hence simple skin-wounds bleed less than those in mucous membranes. Hæmorrhages from the capillaries alone cease spontaneously if the tissue be healthy, because the openings of the vessels are compressed by contraction of the wounded tissue. In diseased parts, which do not contract, even hæmorrhage from dilated capillaries may be very considerable.

Hæmorrhage from the *arteries* is readily recognized, on the one hand, because the blood flows in a stream, which sometimes clearly shows the rhythmical contractions of the heart; on the other, by the bright-red color of the blood. If there be impaired respiration, this bright-red color may change to a dark hue; thus, in operations on the neck, performed to prevent threatening suffocation, and in deep anæsthesia, dark or almost black blood may spurt from the arteries. The amount of blood escaping depends on the diameter of the totally-divided artery, or on the size of the opening in its wall. You must not, however, believe that the stream of blood corresponds exactly to the size of the artery; it is usually much smaller, for the calibre of the artery generally contracts at the point of division; only the larger arteries, such as the aorta, carotids, femoral, axillary, etc., have so little muscular fibre that they contract, in their circumference at least, to a scarcely perceptible extent. In very small arteries, this contraction of the cut vessel has such an effect that, from the increased friction, the blood flows from them without spurting or pulsating; indeed, in very small arteries, this friction may be so decided that the blood flows with difficulty and very slowly, and soon coagulates, so that the hæmorrhage is arrested spontaneously. The smaller the diameter of the arteries becomes, from diminution of the amount of blood in the body, the more readily hæmorrhage will be arrested spontaneously, while otherwise it would have to be arrested artificially. Hereafter, you will often have occasion to see in the clinic how freely the blood spurts at the commencement of an operation, and how much less it will be toward the end, even when we cut larger vessels than were at first divided. Thus decrease of the total volume of blood may cause spontaneous arrest of hæmorrhage; the weaker contractions of the heart have also some influence in this. Indeed, in internal hæmorrhages that we cannot reach directly, we employ rapid abstraction of blood from the arm (venesection) as a hæmostatic; in such cases the artificial excitement of anæmia is not unfrequently the only remedy we have for internal hæmorrhage, paradoxical as this may seem tc you at the first glance. Hæmorrhages from incised wounds of the large arteries of the trunk, neck, and extremities, are always so considerable that they absolutely require to be arrested, unless the openings in their walls be very small. But, when the terminal branch of an artery is ruptured without a wound of the skin, the hæmorrhage may be arrested by pressure on the surrounding soft parts; such injuries subsequently induce other changes, to which your attention will be called under other circumstances.

Hæmorrhage from the veins is characterized by the steady flow of dark blood. This is especially true of small and middle-sized veins. These hæmorrhages are rarely very profuse, so that, in order to obtain a sufficient quantity on letting blood from the subcutaneous veins of the arm at the bend of the elbow, we must obstruct the flow of blood to the heart. If this were not done, blood would only flow from this vein at the time of puncture, further hæmorrhage would cease spontaneously, unless kept up by muscular contractions. This is chiefly because the thin walls of the veins collapse, instead of gaping, as the arteries do when divided. Blood does not readily flow back from the central end of the vein, on account of the valves; we rarely have any thing to do with the valveless veins of the portal system.

Hæmorrhage from the large venous trunks is always a dangerous symptom. Bleeding from the axillary, femoral, subclavian or internal jugular, is usually quickly fatal, unless aid arrive immediately; wounds of the vena anonyma may be regarded as absolutely mortal. The blood does not flow continuously from these large veins, but the flow is greatly influenced by the respiration. In operations about the neck I have frequently seen patients live after their internal jugular vein had been wounded; during inspiration the vessel collapsed so that it might have been regarded as a connective tissue string; during expiration the black blood gushed up as from a well, or still more like the bubbling up of the water from a deep spring.

In these veins near the heart, besides the rapid loss of blood, there is another element that greatly increases the danger; this is the *entrance of air into the veins and heart*, as occasionally takes place with a gurgling noise, on deep inspiration, when the blood rushes toward the heart; this may cause instant death, though not necessarily. I cannot now enter more explicitly into this very remarkable phenomenon, whose physiological effect has not, as it seems to me, been satisfactorily explained; you will again have your attention called to this subject by the books and lectures on operative surgery. I shall merely mention that, on opening one of the large veins of the neck or the axillary vein, there may be a perceptible gurgling sound; the patient instantly loses consciousness, and can rarely be restored to life by instantaneous resort to artificial respiration, etc. Death is probably caused by the entrance of air-bubbles, which press forward into the medium-sized pulmonary arteries, and are there arrested, and prevent further access of blood to the pulmonary vessels.

I have never met any thing of the kind, although I have seen air enter the internal jugular vein, and frothy blood then escape; this had no perceptible effect on the state of the patient. Different animals appear to be susceptible, to various extents, to the entrance of air into the vessels; if we throw only a little air into the jugular vein of a rabbit it dies; while we may sometimes throw several syringefuls into dogs without observing any effects.

Besides the above varieties of hæmorrhage, we distinguish the so-called *parenchymatous hæmorrhage*, which is sometimes incorrectly identified with capillary hæmorrhage. In the normal tissue of an otherwise healthy body, parenchymatous hæmorrhages do not come from the capillaries, but from a large number of small arteries and veins, which from some cause do not retract into the tissue and contract, and are not compressed by the tissue itself. Bleeding from the corpus cavernosum penis is an example of such parenchymatous hæmorrhages, which also occur from the female genitals and in the perineal and anal regions, as well as from the tongue and spongy bones. These parenchymatous hæmorrhages are especially frequent from diseased tissue; they also occur after injuries and operations, as so-called *secondary hæmorrhages;* but we shall speak of these hereafter.

One other point we must refer to here: this is, that there are persons who bleed so freely from a small, insignificant wound, that they may die of hæmorrhage from a scratch of the skin, or after extraction of a tooth. This constitutional disease is called a *hæmorrhagic diathesis;* people affected with it are called *hæmophilen.* The cause of this disease is probably abnormal thinness of the arterial walls; this is congenital in most cases, but may probably result gradually from morbid degeneration and atrophy of the vascular tunics. This frightful malady is usually hereditary in certain families, especially among the males, the females being less liable to it. In these persons hæmorrhage is caused not only by wounds, but light pressure may induce subcutaneous bleeding, spontaneous hæmorrhages, as from the gastric or vesical mucous membrane, which may even prove fatal. It is not exactly in large wounds where medical aid is called at once or very soon, but more particularly in slight wounds, that continued hæmorrhages occur in such persons which are difficult to arrest, partly, as we above stated, on account of slight contractility or total lack of muscular tissue in the vessels, partly on deficient power of coagulation in the blood. It is true, the latter point has not been proved from the blood that escaped, for in the cases where attention was directed to this point the blood flowed like that of a healthy person.

I shall also call your attention to some peculiarities in hæmorrhages from certain localities, especially from those in the *pharynx*, *posterior nares*, and *rectum*, although, strictly speaking, this comes in the domain of special surgery. Wounds of the pharnyx or posterior nares, made through the open mouth by accident, are rare, but, as a result of constitutional disease, we may have very severe spontaneous hæmorrhage from these parts, or these may result from operations, for we not unfrequently have to use knives and scissors here, or to tear out tumors with forceps. The blood does not always escape from the mouth and nose, but it may run down the pharynx into the œsophagus without being perceived. The general effects of rapid loss of blood come on rapidly, which we shall soon describe more minutely, but we are unable to discover the source of the bleeding, which may be behind the soft palate. The patient soon vomits, and at once throws up large quantities of blood; when this ceases there is another pause, and the patient, perhaps also the surgeon, thinks the hæmorrhage has ceased, till more blood is vomited, and the patient grows still weaker. If the surgeon does not recognize these symptoms and apply proper remedies, the patient may bleed to death. I remember one case where several physicians gave various remedies for vomiting of blood and gastric hæmorrhage after a little operation in the throat, and the source of the bleeding was finally recognized by an experienced old surgeon, who arrested it by local applications, and thus saved the life of the patient.

The same thing may happen in hæmorrhage from the rectum. From an internal wound the blood flows into the rectum, which is capable of enormous distention; the patient has a sudden desire to stool, and evacuates large quantities of blood. This may be repeated several times, till the rectum, irritated by the expansion, either contracts and thus arrests the hæmorrhage, or till it is finally checked artificially.

A rapid excessive loss of blood induces changes in the whole body, which are soon perceptible. The face, especially the lips, becomes pale, the latter bluish, the pulse is smaller, and at first less frequent. The bodily temperature sinks most perceptibly in the extremities; the patient, especially when sitting up, is subject to fainting-spells, dizziness, nausea, or even vomiting, his eyes are dazzled, and he has noises in the ears, every thing appears to whirl around; he collects his strength to hold himself up, he becomes unconscious, and finally falls over. These symptoms of syncope we refer to rapid anæmia of the brain. In a horizontal posture this soon passes off. Persons often fall into this state from very slight loss of blood, occasionally more from loathing and aversion to the flowing blood than from weakness. A single fainting of this kind is no measure of the amount of blood lost; the patient soon recovers his forces.

Should the hæmorrhage continue, the following symptoms appear sooner or later: the countenance grows paler and waxy, the lips pale blue, the eyes dull, the bodily temperature is lower, the pulse small, thready, and very frequent, respiration incomplete, the patient faints frequently, constantly grows more feeble and anxious; at last he remains unconscious, and there is twitching of the arms and legs, which is renewed by the slightest irritation, as by the point of a needle, etc.; this state may pass into death. Great dyspnœa, lack of oxygen, is one of the worst signs, but even here we should not hesitate; we can often do something even after apparent death. Young women especially can bear enormous loss of blood without immediate danger to life; you will hereafter have occasion to witness this in the obstetrical clinic. Children and old persons can least bear loss of blood; in young children the results of the application of a leech are often evident for years by a very pallid look and increased excitability. In very old persons great loss of blood, if not immediately fatal, may induce obstinate collapse, which after days or weeks passes on to death; this is probably because the loss of blood is immediately supplied by serum, and in old persons the formation of blood-corpuscles goes on slowly; the greatly-diluted blood proves insufficient to nourish the tissues, whose nutrition is at any rate very sluggish.

When the patient comes to himself after severe hæmorrhage, he has excessive thirst, as if the body were dried up, the vessels of the intestinal canal greedily take up the quantities of water drunk; in strong, healthy persons, the cellular constituents of the blood are quickly replaced, it is true we do not exactly know from what source; after a few days, in a person otherwise healthy, we can perceive few signs of the previous anæmia; soon, too, his strength has recovered from the exhaustion.

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## LECTURE III.

Treatment of Hæmorrhage.—1. Ligature and Mediate Ligature of Arteries.—Torsion.—2. Compression by the Finger; Choice of the Point for Compression of the Larger Arteries.—Tourniquet.—Acupressure.—Bandaging.—Tampon.—3. Styptics.—General Treatment of Sudden Anæmia.—Transfusion.

Gentlemen: You now know the different varieties of hæmorrhage. Now, what means have we for arresting a more or less severe bleeding?

The number is great, although we use but few of them—only those that are the most certain. Here you have a field of surgical operation where quick and certain aid is required, so that the result must be unfailing. Still, the employment of these remedies requires practice; cool-blooded quiet, absolute certainty, and presence of mind, are the first requisites in dangerous hæmorrhage. In such circumstances a surgeon may show of what metal he is made.

Hæmostatics are divided into three chief classes: 1. Closure of the vessel by tying it—ligation. 2. Compression. 3. The remedies that cause rapid coagulation of blood, styptics (from στύφω, to contract).

1. The ligature may be applied in three ways, viz., as ligature of the isolated bleeding vessels, as mediate ligature of the latter with the surrounding soft parts, or as ligation in the continuity, i. e., ligation of the vessel at some distance from the wound.

These varieties of ligation apply almost exclusively to arrest of arterial hæmorrhage. Venous hæmorrhages rarely require ligation—it is only occasionally indicated in the large venous trunks; we avoid it whenever we can, as its results may be dangerous. We shall hereafter inquire in what this danger consists, and at present speak only of the ligation of arteries.

Let us suppose the simplest case; a small artery spurts from a wound: you first seize the artery, as much isolated as possible, best transversely, between the branches of a sliding forceps; then fasten the slide, and the bleeding is stopped. The sliding forceps are best made of German silver, as it rusts less readily than iron. There are many different varieties of these forceps, which are all so arranged that when closed they remain fixed in that position; the mechanism accomplishing this closure varies greatly; the more simple it is, the better. It is interesting to follow the phases of development of this instrument since the days of *Ambrose Paré*, before it attained its present simple completeness. Of late small spring clamps are not unfrequently employed to compress the bleeding arteries; these are very serviceable, if strongly made. Besides these pincettes, we may also use small curved sharp hooks (*Bromfield's* artery-hook) to draw out the artery, but this is not so good a way, for of course the blood would continue to spurt during the subsequent ligation.

Having seized the artery securely, the next thing is to close it permanently; this is done by the ligature. But satisfy yourself first that you have not included a nerve with it, for the coincident ligation of a nerve may not only induce continued severe pain, but even dangerous general nervous affections. For ligating arteries we use silk thread of various thickness, according to the size of the artery; it must be good, strong silk, so that it shall not break when firmly tied; and it should not readily absorb fluids. Have the forceps, which hang from the end of the artery, held up, then from below place the silk around the artery, making first a simple knot and tying it tightly just in front of the forceps, then tie a second knot. Now loosen the forceps; if the ligature is rightly applied, the bleeding must be arrested. The tightening of the knot must be accomplished by pushing the silk forward and stretching it with the points of both fingers. If the silk be good, two simple knots, one over the other, will suffice. When the ligature is firmly applied, cut one end off short and lead the other out of the wound the shortest way. In from 6 to 10 days these can usually be removed. When you propose to close the wound entirely, it is best to use catgut made pliable by soaking in oil; the knots and loops are gradually absorbed, and only rarely thrown off by suppuration.

It is not always possible to take up the spurting artery and ligate it by itself; occasionally it contracts so strongly into the tissue, especially into the muscles or dense cellular tissue, that its isolation is impracticable. Under such circumstances it is difficult to complete the ligation securely; we are very apt to include the blades of the forceps in the ligature, as it is difficult to push the ligature far enough forward. Such cases are proper ones for mediate ligation. After having pulled forward the bleeding part with forceps or a hook, pass a curved needle, held in a needle-holder, around the artery, then tie the ligature so as to encircle the entire end of the artery; tie the knots tightly, as above directed; thus, while closing the mouth of the artery, you will enclose some of the surrounding tissue. Mediate ligation is only to be regarded as an exceptional proceeding, for the ligated tissue dies or the ligature suppurates through very slowly, so that the separation of the latter is much impeded; of course we must guard against including any visible nerve-trunk near the artery in the ligature. In the percutaneous mediate ligation of *Middeldorpf*, we proceed even more summarily; we pass a strongly-curved large needle through the skin, under and across the bleeding artery, and again out through the skin; the thread is tied, and, besides compressing other parts, compresses the artery; the thread remains two or three days. I do not recommend this method; it should only be employed in cases of necessity, and as a provisional hæmostatic.

Whenever the bleeding artery can be seen in the wound, the hæmorrhage is to be arrested by ligature; but, in those cases where the arteries of the periosteum or bone spurt out blood, ligature is impossible, and other methods, such as compression, come into play.

If you have to deal with large bleeding arteries, the proceeding is just the same, only you must be doubly careful in isolating the artery: seize the bleeding end and scrape back the surrounding tissue with a small scalpel, then ligate carefully and accurately; in most cases, when you have the central and peripheral ends exposed in the wound, you should ligate both, for the anastomoses in the arterial system are so free that, if the peripheral end does not bleed at once, it may do so later.

The wound from which a copious hæmorrhage comes may be very small, as a punctured or gun-shot wound. From your anatomical knowledge you should know what large vessel may be injured by such a wound. If, from the free hæmorrhage or its frequent recurrence after compression, you are satisfied that ligation is the only certain remedy for the bleeding, you have the following alternatives: either enlarge the existing wound by careful, clean incisions, and seek for the vessel in the wound while the artery is compressed above, and ligate the divided ends of the artery; or else, while you have the bleeding vessel compressed in the wound, you seek the central part of the vessel above the wound, and then ligate in the continuity. Both operations demand accurate anatomical knowledge of the positions of the arteries, and practice. Which of these two operations you shall choose depends on how you can soonest prudently attain your object, and on which of them will require the smaller new wound. If you think you can expose the artery in the wound without enlarging it much, choose this method as the more certain; but if you consider this very difficult, if at the seat of the wound the artery lies deep under muscles and fascia, especially in very muscular or fat persons, make a regular ligation of the artery above (toward the heart from) the wound.

I shall not here discuss the points chosen after years of trial, on theoretical and practical grounds, for the ligation of arteries. In operative surgery, in the text-books on surgical anatomy, and especially in the operative course, you will be instructed on this point, and must attain practice in certainly finding, neatly exposing, and carefully ligating, the artery, in doing which, you cannot accustom yourself to too much pedantry and technicality.

Although the value of the ligature is recognized by all surgeons of the present day, still attempts have been constantly made to find simpler substitutes which should be just as safe. Some have considered it (unjustly, as it seems to me) a great evil to leave in the wound a silk thread and a portion of ligated vessel to die and become putrid. I pass over the attempts and proposals made for allowing the ligature to heal in the cicatrix, and merely mention *torsion* of the bleeding artery as a mode of closing the vessel mechanically till its walls grow together. The bleeding vessel is seized with strong, accurately-closing forceps, drawn forward half an inch, and twisted on its axis five or six times; I usually draw it out as far as possible, and twist till it breaks off. In this way I have twisted vessels from the smallest size to that of the brachial, so as to securely arrest the bleeding. If branches leave the artery just above the bleeding-point, it will not be movable enough to make the torsion securely; hence I have never tried torsion for the femoral; but other surgeons have done so successfully.

2. *Compression.*—Pressure on the bleeding vessel with the finger is such a simple, apparent method of arresting hæmorrhage, if we may call it a method, that it is strange the laity do not resort to it at once; any person that has seen one or two operations would instinctively hold his finger on the bleeding vessel; still how rarely people do this in a case of accidental wound! They prefer resorting to all sorts of home remedies; spider-webs, hair, urine, and all sorts of filth, are smeared over the wound, or else they run for some old woman who can arrest the bleeding by magic. And no one around thinks of compressing the wound.

Methodical compression may be made for one of two purposes, as provisional or permanent.

*Provisional* compression, which is used till we can determine how the bleeding may be best arrested permanently, may either be made by pressing the bleeding vessel in the wound against a bone, if possible, or by pressing the central part of the artery against the bone at some distance from the wound; the former, as we have already stated, is to be done when we propose to ligate the trunk; the latter, when we wish to tie the bleeding end of the artery, or to examine the wound more carefully.

Where shall we compress the artery, and how shall we do it most effectually? To compress the right *carotid*, you would place yourself behind the patient, and lay the tips of the second, third, and fourth fingers of the right hand along the anterior border of the sterno-cleido-mastoideus muscle, about the middle of the neck, and press firmly against the spine, while you pass the thumb around the neck, and with the left hand bend the patient's head gently to the wounded side and somewhat backward. You should distinctly feel the pulsation of the carotid artery. Firm pressure here is quite painful for the patient, for the vagus nerve is unavoidably compressed, and the tension of the parts necessarily acts on the larynx and trachea. From the free anastomoses of the two carotids, the effect of compression of one of them, in arresting bleeding from an artery of the head or face, is not generally very great, and perfect compression of both vessels requires so much space, that we must generally be satisfied with diminishing the volume of the arteries by incomplete compression. Compression of both carotids is always a very painful and terrifying operation for the patient, especially on account of the strong secondary pressure made on the larynx and trachea; hence it is rarely employed.

Compression of the *subclavian artery* may be more frequently required, especially in wounds of this artery in *Mohrenheim's* fossa and in the axilla. In this operation also you may best stand behind the recumbent or half-sitting patient; with your left hand incline the head of the patient toward the wounded (right) side, and push your right thumb firmly behind the outer border of the clavicular portion of the relaxed sterno-cleido-mastoid muscle, so that you may firmly compress the artery against the first rib, at the point where it passes forward between the scaleni muscles. Here also pressure is painful, from the liability of the brachial plexus of nerves to be included in the compression; still, by employing sufficient force, we may completely compress the artery so as to arrest pulsation of the radial. But the thumb soon grows tired and loses sensation; hence various aids have been devised—instruments by which the compression may be made certainly. One of the most convenient means is a short thick key whose wards are wrapped in a handkerchief and the handle held firmly in the palm of the hand; you place the wards of the key over the artery, and compress it firmly against the first rib. But this cannnot fully replace compression by the finger of a skilled assistant, for with the instrument you of course cannot feel if the artery slides away from the pressure.

From its position the *brachial artery* may of course be readily compressed; in doing this, you place yourself on the outer side of the arm, take the arm in your right hand, so as to lay the second, third, and fourth fingers along the inner side of the belly of the biceps, about the middle of the arm or a little above it, surround the rest of the arm with the thumb, and press against the humerus with the fingers; the only difficulty here is to avoid simultaneous compression of the median nerve, which at this point almost covers the artery. By compressing the brachial artery, we may readily arrest the radial pulse, and we may employ this compression with great advantage if we desire to ligate either the radial or ulnar artery on account of wounds, or to amputate at the forearm or the lower part of the arm.

In hæmorrhages from the arteries of the lower extremities we compress the *femoral* artery at its commencement, that is, immediately below *Poupart's* ligament. Here, where it lies just in the middle between the tuberculum pubis and anterior inferior crest of the ileum, the artery should be pressed against the horizontal branch of the pubis. The patient should be recumbent; compression should be made with the thumb, and is easy, because at this point the artery is superficial. As far down as the lower third of the thigh, the femoral artery may be compressed against the femur, but this can only be done certainly by the finger in very thin persons; in most cases we employ for this purpose a special compress called a tourniquet.

By a *tourniquet* we mean an apparatus by which we press an elongated oval piece of wood or leather, a pad, against an artery, and this against the bone, by means of a twisting, screwing, or buckling mechanism. Since a long compression of the brachial or femoral arteries is very fatiguing, we may advantageously call it to aid in compressing these arteries. The form of instrument that we now employ is the screw tourniquet of *Jean Louis Petit.* The pad, which is movable on a band, is to be applied exactly over the point corresponding to the artery, and opposite the screw, under which a few folds of linen are to be placed, to prevent too great pressure on the skin. Then buckle the band around the extremity, and by means of the screw and band draw the pad tighter till the subjacent artery ceases to pulsate. In an amputation-wound, if we do not at once see the mouth of the artery, we may loosen the screw slightly and permit a little blood to escape from the artery, which at once shows its position; then screw up the tourniquet at once, and ligate the artery. This is the great advantage of the screw. When the apparatus is well made and carefully applied, it is of excellent service. It is true, the band around the limb unavoidably compresses the veins, especially the subcutaneous veins; nevertheless, on account of the pad, it acts chiefly on the artery. With a piece of broad bandage and a round block of wood, or a roller of bandage and a short stick, you may readily improvise such a tourniquet; still, if this improvised apparatus does not secure the artery very firmly and securely, I should advise more certain modes of compression, of which I shall speak immediately. The facility of checking even considerable hæmorrhages by means of the tourniquet, might delude us into leaving it on for a long while, until the bleeding stopped of itself, and we should thus escape the trouble of ligating. This would be a great error. If the tourniquet remains on half an hour, the extremity below it grows blue, swells, loses sensation, and circulation in the part may be entirely arrested, and it will die; through your whole life you would blame yourself for such an error, which might greatly endanger the life of your patient.

Hence, application of the tourniquet is only admissible as a provisional hæmostatic. It is almost impracticable to compress a large artery with the finger till the hæmorrhage shall be certainly arrested spontaneously. Still, cases may arise where compression with the finger is the only certain mode of arresting bleeding from smaller arteries, as in hæmorrhages from the rectum or deep in the pharynx, when other means have failed; here, compression with the finger must sometimes be continued half an hour to an hour, or longer, for ligation of the internal iliac in the former case, and of the carotid in the latter, are as dangerous as they are uncertain for a permanent arrest of the bleeding.

To avoid the danger from venous congestion after constricting the limb, before applying the tourniquet we may apply a bandage firmly to the extremity from below upward, and thus press the blood out from the limb. Formerly this procedure was applied to limbs about to be amputated, and thus the hæmorrhage was reduced to a minimum. *Grandesso Silvestri*, a physician of Vicenza, recommended elastic bandages for this purpose, and instead of the tourniquet applied a thick elastic tube several times around the limb. In ignorance of this advice, which was little known, *Esmarch* resorted to the same method, and called attention to the great benefits derived from it; since when it has become very popular. In fact, by means of this appliance long operations may be done without loss of blood. The extremities may be rendered bloodless and kept so for an hour without injuring their vitality. After ligating all the visible vessels, the elastic tube is to be loosened, and the blood will again enter the vessels; if any more then permit the escape of blood, they may be ligated. This method of making a limb bloodless and keeping it so is a great advance in modern surgery; and by this means operations may be done which without it we should not have dared to attempt.

[The bleeding which follows removal of the elastic tube is the only objection to its use. Attempts have been made to prevent it by applying a strong current of electricity to the wound before removing the tube, but this is not reliable. It is better, instead of applying a bandage, to hold the limb vertical for a time, then put on the tube. The limb is not rendered so bloodless by this means, but after the tube has been removed there is less bleeding.

Forced compression by means of spring forceps has been used by *Koeberle* and *Verneuil*, especially where the artery was not very accessible. The bleeding vessel may be isolated or seized with surrounding tissues, the forceps being left in place a few minutes for small vessels, and for larger ones as much as twenty-four hours, when the calibre will be occluded by coagulum. This procedure is good in some cases, but cannot replace the ligature. *Vanzetti's* uncipressure depends on the same principle: where there is bleeding from a deep wound, traction with a hook in a certain direction compresses the vessel by the tension and arrests the hæmorrhage.

A very important method of arresting hæmorrhage is compression of the bleeding vessels by pressure on the skin or the wounded surface; when circumstances are favorable, that is, when the bleeding vessel can be pressed against some firm body, as a bone, bleeding may always be arrested in this way. On the same principle we may try forced flexion of limbs to an acute angle; of course, this is only applicable to the extremities. If we flex the elbow or knee to the maximum, the pulse disappears below the joint from compression of the artery. This operation has been used for hæmorrhage as well as for aneurisms, but from the simultaneous compression of the nerves it is very painful, and cannot be very long continued.

Cold has long been used as a hæmostatic, and now *heat* is applied for the same purpose; this seems paradoxical, as warmth generally induces expansion and relaxation. This is true of moderate degrees of heat, while higher ones are irritating and cause contraction of the tissues and vessels. In hæmorrhages from the uterus injections of hot water are very serviceable, apparently from inducing active contractions of the organic muscular fibres of the uterus; in hæmorrhages from vessels not lying in contractile tissue, as after amputations, irrigation with hot water causes temporary arrest of bleeding, but this soon recurs with greater energy than if the vessels had been left to themselves.

Recently I have tried the Penghawar Djambi a few times, and can testify that when quantities of it are pressed firmly on the wound it acts better than charpie as a styptic; I will not pretend to say that it is better than liquor ferri, but it smears the wound less, even if left on for some days. Penghawar Djambi is the light yellowish, soft, hairy substance from the trunk of the *Cibotium Cuminghii*, a native of the East Indies. [This must closely resemble the styptic cotton so commonly used by us. During the past year, 1878, *Paquelin's* cautery has been used a good deal, and is very convenient. By a hand-bellows a stream of benzine vapor is thrown against a platinum cap which has been previously heated in a spirit-lamp; after it has once reached a red or white heat, it is easily kept at that point by the benzine vapor. It is readily used, and when at a white heat its application is not painful. For hæmorrhages from the vagina or uterus, water at a temperature of 110° to 125° Fahr. may be injected for some time by means of *Davidson's* syringe.]

[In a pure incised or punctured wound one of the symptoms is *extravasation*, that is, an escape of fluid from the tissues; usually we only think of the extravasation of blood, as this is the most apparent. But there are non-vascular tissues, such as the cornea, which do not bleed; and, moreover, in every incised or punctured wound there is an escape of lymph from the interspaces of the skin and connective tissues, which is, however, usually obscured by the bleeding. Opening of physiological cavernous tissues may also allow other fluids to be mixed with the blood. Escape of the fluids of the tissues, especially lymph, requires no particular treatment.]

Quite recently the genial surgeon and obstetrician, *Simpson*, of Edinburgh, whom you already know as the introducer of chloroform, has recommended a method which I cannot recognize as a perfect substitute for ligation, but which is in many cases of practical use; this is the compression of the bleeding artery by a needle—*acupressure.* Acupressure may be made in various ways. For instance, in an amputation-flap, you introduce a long insect, or sewing-needle, nearly vertically through the skin and soft parts to within one-quarter or one-half an inch of the artery; turn the needle horizontally, bring its point close over or under the artery, and at about the same distance from the artery you push it into the soft parts, and pass it out through the skin nearly vertically, so that the artery shall be compressed between the needle and the soft parts, or, still better, against a bone. Should this compression not act perfectly, as it would rarely be likely to in large arteries, if the first needle was applied above the artery, pass a second one below it, and so compress the artery between the two needles, or else press the artery against the needle by means of a wire loop. In amputations I prefer acupressure by torsion; I pass the needle transversely through the artery, which is drawn forward, and with the needle make a half or whole rotation in the direction of the radius of the surface of the flap, until the bleeding is arrested, and then insert the point of the needle into the soft parts. The needles may be removed after forty-eight hours, without renewal of bleeding. The extensive experience of English surgeons in the success of this bold operation first gave me courage to try it, and I must acknowledge that in several amputations, even of the thigh, I have seen no objection to it. I cannot quite believe that acupressure will altogether displace ligation, as *Simpson* prophesied. In this operation, to which I have resorted in most of my amputations for several years, I employ long golden needles with large heads, because other metals rust easily, and silver is too soft, and platinum too expensive.

Quite recently *Von Bruns* has applied small ligature rods, with which loops of silk are applied around and retained against the artery, previously drawn out. These, like acupressure-needles, are removed after forty-eight hours. I have just tried this procedure with perfect success on the femoral artery in an amputation of the thigh.

In venous hæmorrhage, or bleeding from numerous small arteries, especially in so-called parenchymatous hæmorrhage, a regular tampon must be applied, by means of bandages, compresses, and charpie.

If you have a hæmorrhage from the arm or leg, that you wish to arrest by compression—if, for instance, large quantities of blood are being poured out from a dilated diseased vein, or if there be bleeding from numerous small arteries—you may apply a bandage firmly from the lower to the upper part of the extremity, having previously covered the wound with a compress and charpie, and after applying several thicknesses of linen along the course of the chief artery of the extremity. For the latter purpose you may also employ the graduated compress, which you will learn to make in the course on bandages. To this, which is called *Theden's* dressing, it is well to add a splint, to keep the extremity perfectly quiet, for the bleeding is readily renewed by muscular contractions. These graduated compresses, carefully made, are particularly serviceable on the battle-field, in gun-shot and punctured wounds; by their aid we may arrest hæmorrhage from the radial, ulnar, anterior and posterior tibial, and even from the brachial and femoral arteries. In the former or smaller arteries, by leaving the dressing on six or eight days, we may arrest the bleeding permanently, but in the latter it only acts as a provisional hæmostatic; it must be followed by ligation, if we wish to be at all sure of avoiding a recurrence of the bleeding. We may also employ compression in hæmorrhages from the thorax, as in case of parenchymatous hæmorrhage after removal of a diseased breast; here we may dress the wound with compresses and charpie, and retain them in position by bandages around the thorax. But, for such a bandage to be efficacious, it must be very annoying to the patient; on the whole, it is better to ligate the bleeding arteries, even if there should be many of them; by so doing, both you and your patients will be better off, for you will not be worried and disturbed by the secondary hæmorrhages following these operations as a result of hasty ligation and insufficient compression.

In some parts of the body you cannot employ compresses, as in bleeding from the rectum, vagina, or posterior nares. Here the *tampon* (from tampon, plug) is serviceable. There are many varieties of tampons, especially for hæmorrhage from the vagina or rectum. One of the simplest is as follows: Take a four-cornered piece of linen, about a foot square; placing the middle of this over two, three, or five fingers of your right hand, pass it into the vagina or rectum, and fill the space left by the removal of your hand with as much charpie as you can get in, so that the vagina or rectum will be fully distended from within, and thus strong pressure be made on its walls; when the hæmorrhage is arrested, leave the tampon in till the next day, or longer if necessary, then remove it by gentle traction on the linen, which serves as a sac for the charpie. You may also make a ball of charpie or linen by wrapping a string around it, and leave a long string hanging out by which to remove it; as such a tampon may be either too large or too small, I prefer the first method, in which we may fill the linen sac to the extent we desire.

If the bleeding come from the portio vaginalis uteri, after an operation, for instance, a more certain way is to hold back the posterior wall with a large *Sims's* speculum, thus bringing the portio vaginalis into view, and press a tampon firmly against the bleeding part; for it requires an incredible quantity of charpie to fill the vagina of a woman who has borne many children, so that no blood can pass through, and it causes great pain.

In profuse bleeding from the nose, which mostly comes from the posterior part of the inferior meatus, and not unfrequently from the posteriorly-situated cavernous tissue of the lower turbinated bone, plugging the nose from the front proves inefficacious and useless; the bleeding continues, and the blood either passes into the pharynx or flows out of the other nostril, as the patient presses the velum pendulum palati against the wall of the pharynx, and shuts off the upper part of the pharyngeal cavity. Hence, we must be prepared to plug the posterior nares; we may do this by the aid of *Belloc's* sound. This exceedingly convenient instrument consists of a canula about six inches long and slightly curved at one end; in the canula is a steel spring of much greater length, with a perforated button-head at one end. You prepare beforehand a thick plug large enough to fill the posterior nares, and have a thread attached to it. (You may make this plug by laying threads of charpie side by side and tying them tightly together in the middle with a silk thread.) You apply this plug by passing the instrument, with retracted spring, through the inferior nasal meatus, then pushing the spring forward till it appears below the velum in the mouth. Pass the thread attached to the plug through the eye in the head of the spring, tie it there, and draw both canula and spring out of the nose; the thread attached to the latter and the plug fast to this must follow, and if you draw tightly on the thread the plug is pressed firmly into the posterior nares; if the bleeding be now arrested, as it usually is, if the plug (which should not be long enough for its end to reach the larynx) was not too small, you cut loose the thread, leave the plug in till the next day, then withdraw it by the thread left hanging from the mouth; this is usually easily done, as the plug is generally covered with mucus and is consequently smooth. As this instrument is not always at hand, we may use an elastic catheter or a thin slip of whalebone for the same purpose, introducing it through the nose seizing it with the finger behind the velum, and bringing the end out of the mouth to tie the thread to it. But the employment of this substitute requires more dexterity than is necessary for *Belloc's* sound.

3. *Styptics* are remedies which act partly by causing contraction of the tissue, partly by inducing rapid and firm coagulation. The number of remedies recommended is immense; we shall only mention those that have a proved reputation under certain circumstances.

Cold not only irritates the arteries and veins to contract, but also makes the other soft parts contract and thus compress the vessels; the current of blood is gradually more obstructed, and may even stagnate entirely, when the part is completely frozen. It seems to me, however, that the recommendation of cold as a hæmostatic is often carried too far; I advise you not to rely on it too much. Cold may be employed as follows: first, we may squirt ice-water against the bleeding wound, or into the vagina, rectum, into the bladder through a catheter, into the nose or mouth—here the mechanical irritation of a strong stream of water is added to that of the cold; or you may lay pieces of ice on the wound, or introduce them into the cavities, or have them swallowed in gastric or pulmonary hæmorrhage; or, lastly, you may fill a bladder with ice and apply to the wound, to be left on for hours or days.

The *absolute quiet* to be observed in all hæmorrhages and the diminution in size of the arteries as a result of the bleeding that has already occurred, may often have more effect in arresting the hæmorrhage than ice has, while it receives all the credit. I will not dissuade you from using cold in moderate parenchymatous hæmorrhages, but do not expect too much from it in bleeding from large arteries, and do not waste too much time over it, for time is blood—blood is life.

The same is true of the common local remedies, vinegar, solution of alum, etc., which also contract the tisues and thus compress the vessels; they are very good for arresting capillary hæmorrhages from the nose, but you must not expect any thing wonderful from them.

The *hot iron*, ferrum candens, causticum actuale, acts by charring the ends of the vessels and the blood, and the escape of the blood is arrested by the resulting firm slough. You only need to hold a rod of iron with a wooden handle at one end, and at the other a small iron head heated to a white heat, close to the bleeding spot, to form a black crust instantly; indeed, the tissue occasionally blazes up even from the radiated heat. A red-hot iron pressed on the bleeding spot has the same effect, but is apt to cling to the resulting eschar and pull it off again. This iron rod (cautery iron) is usually heated to the proper degree in a furnace with bellows. Under some circumstances the hot iron may be very convenient for arresting hæmorrhage; formerly, before ligation was known, it was the most celebrated styptic. The Arabian surgeons usually heated their amputating knives red hot, a proceeding that even *Fabricius Hildanus* extolled, although he preferred burning the bleeding arteries separately with fine-pointed cauteries, in which he must have had an enviable expertness.

Quite recently a similar method has been invented, namely, the use of platinum heated by the galvanic battery. This is the so-called *galvano-caustic* introduced into Germany by *Middeldorpf*, which may sometimes be employed with advantage. As you may readily understand, in practice we have not always at hand an iron properly shaped for arresting hæmorrhage, such as you see in the surgical clinics. *Dieffenbach*, the most talented German operator of this century, who was at the same time a most original man, once, lacking other means, being alone in a poor dwelling, arrested a hæmorrhage following the extirpation of a tumor from the back, by means of the tongs which he heated in the stove. A knitting-needle, stuck in a piece of wood or a cork, and heated at the lamp, may answer the purpose of the hot iron.

A remedy which not only equals, but occasionally surpasses, the hot iron in its effects, is *liquor ferri sesquichlorati;* this forms with the blood such a leathery, adherent coagulum, that it acts excellently as a styptic. To apply it, you press a piece of charpie, moistened with it, firmly against the wound; after having washed off the blood with a sponge, hold it there from two to five minutes; you will thus be able to arrest quite free arterial hæmorrhage. If the first application does not succeed, try it a second or third time; this remedy will rarely fail you; but it makes a slough, behind which there is often sanious suppuration mixed with gas-bubbles; hence we should not employ this styptic needlessly.

The application of *punk* and blotting-paper to bleeding wounds is an old popular remedy; the punk sticks fast to the blood and the wound, if the bleeding be not excessive; in hæmorrhages at all free it is useless without simultaneous compression; occasionally it is very efficacious, and is highly praised by some surgeons. Dry charpie pressed firmly on the wound has the same effect, according to my experience.

Other hæmostatics are *oil of turpentine* and *aqua Binelli*, in which the creosote is chiefly efficacious; concerning the former alone have I any experience, and I recommend it strongly; when I studied in Göttingen, it was also specially recommended by my preceptor, *Baum*, and I used it once with such striking benefit in a doubtful case that I have a certain devotion for it. It is, however, an heroic remedy, not only because application of turpentine-oil to a wound induces severe pain, but also because it excites severe inflammation in the wound and its vicinity. I will relate the case where I employed it.

A young, feeble woman suffered, after confinement for many months, from an extensive suppuration behind the right breast, between the mammary gland and the fascia of the pectoral muscle; numerous incisions had already been made through the breast, and about its circumference, to give free access to the pus which formed in such quantities; but the openings soon closed again, and new ones had to be made, as the wound did not heal from below. From one such incision, which I made quite extensive, severe hæmorrhage resulted, blood welled up from the depth of the abscess, and I was unable to find the bleeding vessel; it flowed continuously, as if from a spring. First, I filled the cavity with charpie and applied a bandage; the blood soon oozed through this dressing; I removed it and injected ice-water into the various openings; the bleeding moderated. I again made firm compression, and the hæmorrhage seemed arrested. I had scarcely reached my room in the hospital when I was called by the nurse, because the blood again oozed through the dressing; the patient had fainted, was pale as a corpse, and the pulse was very small. The bandage had to be removed at once. I now thrust pieces of ice through the different openings into the cavity under the breast; still the bleeding was not arrested. The patient went from one fainting-fit into another, the bed flowed with blood and ice-water, the patient lay unconscious, with cold limbs and upturned eyes, the nurses constantly trying to resuscitate the patient by holding ammonia to the nose, and rubbing the forehead with Cologne water. At the commencement of my surgical life, unaccustomed to quiet and presence of mind in such scenes, caused by my own act, I shall never forget this situation. I thought it would be absolutely necessary to amputate the breast at once, to find and ligate the bleeding artery, but determined to make one more attempt with oil of turpentine. I soaked a few wads in this substance, introduced them into the wound, and the bleeding was instantly arrested. The patient soon revived; the turpentine, which was left in twenty-four hours, caused intense reaction in the abscess cavity, whose walls became detached. Subsequent active granulation induced in three weeks a cure which had for months been patiently and perseveringly sought in vain by physician and patient. I cannot explain to you how bleeding is arrested by oil of turpentine and creosote; they do not cause particularly firm coagulation of the blood; probably the intense irritation they induce excites a peculiarly energetic contraction of the divided capillaries.

You will rarely see styptics employed in the surgical clinic; they are rather favorites of the practising physician, who is not accustomed to ligate arteries. Where we can ligate or compress, we should not use styptics. In parenchymatous bleeding from the face, neck, or perinæum, we may resort to styptics with advantage, if it makes no difference whether the wound suppurates subsequently; but, if the hæmorrhage be considerable, and styptics fail, subsequent ligation is much more difficult, as the wound is often terribly smeared up by the previous applications.

In surgery you have nothing to expect from the internal administration of remedies recommended as styptics. Absolute quiet, keeping cool, narcotics, purgatives, may occasionally be of great assistance in congestive hæmorrhages, but their action is far too slow for the bleeding that we have to deal with in surgery.

The general debility from profuse hæmorrhage will, of course, be most effectually combated by arresting the bleeding; but, while doing this, you may have the assistants, not otherwise employed, try to resuscitate the patient by smelling-salts, sprinkling with water, etc. You should not yourself join in these attempts, till the bleeding is stopped; then you may give wine, rum, or brandy, warm coffee, or soup; cover the patient up warmly; let him take a few drops of spirits of ether or acetic ether, and smell ammonia, etc. I have never had a patient bleed to death under my hands, but have met two cases where the patients died, two and five hours after extensive operations, with dyspnœa and spasmodic contractions, apparently as a result of the great loss of blood; these cases decided me, under similar circumstances, to inject the blood of a healthy person into the veins of the bleeding one. This operation, which is called *transfusion*, is quite ancient; it originated in the middle of the seventeenth century. After the world had been for a time astonished at its boldness, it was laid aside and derided, but, toward the end of the last century, it was again drawn from the shade of oblivion by English physicians, especially the obstetricians. After *Dieffenbach* had made some attempts again to introduce transfusion into Germany without success, *Martin* has of late the credit of again calling attention to it as a mode of saving life, while *Panum* has exhaustively treated the subject in physiological experiments. Statistics show that the operation was favorable in the great majority of cases, and was very easy to perform. Although formerly lamb's blood was successfully injected into man's veins, it is best and most natural to choose blood from a young, healthy, and strong human being. The instruments required are a knife, forceps, scissors, a fine canula, and a 4–6 oz. glass syringe to fit it. We open the vein of a healthy, strong young man, in the manner hereafter to be described, and receive first about four ounces of the blood in a rather high bowl, standing in a basin full of blood-warm water; the blood, flowing into the bowl, is beaten with a twirling stick, till the fibrine is separated. While this is being done, the most perceptible subcutaneous vein at the bend of the elbow of the patient is to be exposed by an incision through the skin; then two silk threads are to be passed under it, the lower one is drawn on without closing it, so that no blood may escape by the subsequent fine oblique incision made in the vein by the scissors. The canula is passed up into the now gaping opening in the vein, and the upper thread is crossed over it without being tied; some blood should escape through the canula, so as to fill it and drive out the air. Meanwhile, the assistant has completed the venesection and filtered the whipped blood through a fine cloth; then the previously-warmed syringe is to be filled with the blood, inverted and the air forced out, placed firmly in the canula, and the blood injected very slowly. Experience has taught that it is not advisable to inject more than four to eight ounces of blood, and that this is enough to recall life. We should never empty the syringe entirely, and cease at once if the patient has dyspnœa. When the injection is completed, we remove the ligatures and canula, and treat the wound as after venesection. There has been much dispute, as to whether or not it is necessary to remove the fibrine from the blood to be injected. *Panum's* experiments have clearly proved that fibrine is *not* necessary in resuscitation by transfusion, and that, even with the greatest care, it may act injuriously by clotting. The active element in this operation appears to be the introduction of blood-corpuscles as bearers of oxygen. Possibly, transfusion has a still wider future; at all events, it might be worth while to try it in excessive anæmia, resulting from other, sometimes unknown, causes, even although, according to *Panum's* excellent observations, the blood itself does not nourish, but is only the bearer and forwarder of nourishment. The experiments made by *Neudörfer*, during the last Italian War, on the wounded who had become anæmic from profuse suppuration, had no brilliant results, it is true, but further trials should be made of this operation, which with proper care is not dangerous.

*Hueter* has studied transfusion most thoroughly of late; he recommends injecting beaten and filtered venous blood into an artery (such as the radial or posterior tibial) in a peripheral direction, just as was once done by *Von Graefe*. As *Hueter* has demonstrated that this arterial transfusion is easier than the venous, it deserves the preference, because by it we avoid the danger of pulmonary emboli. No abnormal symptoms occurred where *Hueter* operated on the hands and feet; but I doubt if it would often be possible to introduce a canula into these small arteries in a patient bleeding to death; in such a case we should have to choose the brachial artery.

The enormous increase of bodily temperature, the occurrence of bloody urine, and other symptoms, following this operation, show that it has a very decided influence on the physiological action of the organism. As this operation has always been performed in vain by myself and my assistants, I am much less in favor of it than formerly, when I only knew it from the accounts of others.

I cannot here enter on the treatment of the later results of considerable hæmorrhages; it will be evident to you that, in general, the chronic effects, the deficient formation of new blood, must be combated by strengthening and nourishing diet and medicines.

## LECTURE IV.

Gaping of the Wound.—Union by Plaster.—Suture; Interrupted Suture; Twisted Suture.—External Changes perceptible in the United Wound.—Healing by First Intention.

After entirely arresting the hæmorrhage from a wound, cleaning its surface with cold water, and satisfying yourself of its depth, and of the character of the parts divided, in doing which you must notice whether a joint, or one of the cavities of the body, has been opened, a large nerve divided, or a bone exposed or injured, etc., you will turn your attention to the third symptom in the fresh wound, that is, its gaping. On division skin, fascia, and nerves will separate, partly from their own elasticity, partly because they are attached to the muscles, which, from their contractility, shrink together immediately after being divided, and whose cut surfaces, consequently, especially in transverse wounds, are more or less separated.

At first we shall consider only those incised wounds where there has been no loss of substance, but only a simple division of the soft parts. For such a wound to heal quickly, it is desirable that the two edges should be brought exactly together, as they were before the injury; to accomplish this, we make use of strips of adhesive plaster or of sutures.

In wounds where the cutis is scarcely divided, as so often happens in the common incised wounds of the fingers, we may use *isinglass-plaster* with advantage. It consists of a solution of ichthyocolla in water, mixed with a little spirits of wine, painted over a thin, firm silk stuff or paper; the back is often painted with tincture of benzoin, which gives the plaster a pleasant odor. As the plaster readily loses ens under moist compresses, it is often advisable to paint it with collodion, after it has dried.

*Collodion* is a solution of gun-cotton in a mixture of ether and alcohol. If this fluid be painted over the plaster and the skin immediately adjacent, the ether quickly evaporates, and a fine membrane insoluble in water remains, often puckering up the skin. A further therapeutic use may be made of this contractile action of collodion, by painting it on the inflamed skin, either directly, or, still better, after covering the part with a thin, coarse-meshed cotton-cloth (gauze); this causes moderate, even pressure. When you use collodion to fasten the plaster, avoid applying it directly to the wound; this not only causes unnecessary pain, but may also induce inflammation and suppuration of the wound, which should be particularly avoided.

If the cutis be divided, and the plaster must resist any considerable tension in keeping the edges of the wound together, ichthyocolla-plaster proves insufficient, and *adhesive plaster* must be employed. Of this we have two varieties, besides innumerable modifications, from attempts to make it cheaper and better. Emplastrum adhæsivum, emplastrum diachylon compositum, our common adhesive plaster, consists of olive-oil, litharge, resin, and turpentine. While it is fluid from heat it is painted on linen, and it is generally used in strips, which are laid over the wound, and hold its edges together. When fresh, this plaster adheres excellently, but loosens after a time, if moist compresses be applied over it. Very sensitive skins are irritated by this plaster if it is frequently applied; then we may resort to the other adhesive plaster, the *emplastrum cerussæ* (emplastrum adhæsivum album), which is prepared from olive-oil, litharge, and white lead, with hot water. This plaster adheres less firmly, but has the advantage of smearing the lips of the wound less than the yellow plaster. A mixture of equal parts of the two plasters lessens the objections and combines the advantages.

In large wounds we now avoid the use of adhesive plaster more than formerly, and in its place employ the *suture* more commonly. When we wish to unite wounds by the suture, we generally choose between two varieties, the interrupted (sutura nodosa) and the twisted suture (sutura circumvoluta). There is some truth in the assertion that, by the introduction of a foreign body, such as a thread or needle, we maintain constant irritation in the edges of the wound, but this cannot equal the great advantage obtained by the certainty of adjustment of the edges of the wound by means of sutures. Hence, except adhesive plaster, almost all substitutes for the suture, in which ancient and modern surgery has exhausted itself, after being fashion-able for a time, have been thrown aside. The suture has not yet been dropped, and probably never will be, any more than ligation.

There are certain parts of the body, as the scalp, hands, and feet, where we try to avoid sutures, because there certain inflammatory processes, which have often been ascribed to the suture, readily assume a dangerous character; but I think there is a good deal of prejudice in this. Wounds of the head are especially prone to cause inflammations of the skin and subcutaneous tissue; extensive statistics have never shown whether this tendency is particularly increased by the irritation from sutures. There are many articles of faith handed down from preceptor to pupil, from one text-book to another; many of them are a sort of Hippocratic traditions, full of practical truth; to these I pay full respect; others are based on accidental observations and consequent judgments; among the latter, I class the objection to sutures in scalp-wounds. Reviewing my own experience, I remember more cases of inflammation following wounds where no sutures were introduced than where they were. It is very important, however, at once to recognize inflammations beginning in the head, and to remove the sutures. The amount of gaping and the forms of the wound (e. g., a flap-wound or not) at once show the necessity for sutures. One would never take any unnecessary trouble in introducing sutures, unless urged by excess of surgical zeal; but where, for the reasons above given, adhesive plaster will not answer, we should employ sutures.

For the interrupted suture we use surgical needles and silk thread or wire. *Surgical needles* differ from ordinary ones, in having a lance-shaped, ground point, which pierces the skin more readily than the round point of a sewing-needle; they are also of somewhat softer steel than English sewing-needles, so that they do not spring so much. Their thickness and length vary greatly, according as we wish to apply a strong thread deeply where the edges of the wound are tense, or only to use a fine thread to bring the edges together exactly. All needles should, however, have a good-sized eye, so that we may not, like a tailor, lose time in threading them, but do so readily and quickly. The needle may be either straight or curved. The curve should vary with the locality where we wish to sew; for instance, very fine, strongly-curved needles are required for sewing about the inner canthus of the eye; large, strongly-curved needles are needed for sewing up a perinæum, ruptured during labor, etc. The curvature may either be in the whole needle or only at the pointed end; for instance, for certain operations, it is shaped like a fish-hook; the variety is very great. For sewing such wounds as usually present themselves in practice, you need only a few fine and coarse straight and variously-curved needles.

The thread is usually of silk, whose coarseness corresponds to the size of the needle. Formerly I always sewed with the red German silk, which has long been used for this purpose; but in England I found a sort of undyed, strongly-twisted silk, which, even when very fine, is so strong that, with thread as fine as a hair, we may sew up wounds and draw them together. Moreover, this silk imbibes so little moisture that it may lie for days in the wound without swelling or irritating. Now I use only this so-called Chinese silk. Another material for sutures has been lately used in England and America, viz., *silver* or *iron wire*. It must be very fine and soft; the iron wire for this purpose is well annealed. The trial of this material was first induced by the long-known fact that, when metals were introduced under the skin or anywhere in the body, they usually excited no suppuration, but the parts often healed over them. Hence, it was thought that the inflammations often occurring at the points of suture might be avoided by using metal instead of the animal substance silk. In truth, it cannot be denied that this suppuration is less apt to occur from metal than from silk thread, still experiments of *Simon* have shown that the suppuration from sutures depends greatly on the thickness of the thread. From my own experience I can affirm that fine silk threads cause as little suppuration along the course of the suture, and may heal in, just as well as metal ones.

We come now to the *application of the interrupted suture.* You do it as follows: with a toothed forceps you first seize one lip of the wound; pass the needle through the skin, about two lines from the edge, as deep as the subcutaneous tissue, and bring it out through the wound; now seize the other lip of the wound with the forceps and pierce it from the wound up toward the skin, exactly opposite the first point of entrance, then draw the thread through and cut it off, leaving both sides long enough to tie readily in a knot. Now make a simple, or, if the tension of the borders of the wound be great, a surgeon's knot, and draw it tight, seeing that the edges of the wound are in exact apposition; then make a second knot, and cut off both threads, close to the knot, so that no long ends of thread may get in the wound.

Should you desire to use wire, you thread it as you do the silk on the needle, draw a short portion through the eye and bend it, then make the suture as above described. When the wire is very soft, we can tie a knot with it nicely, just as with a silk thread; still, the whole of this manipulation is much less pleasant with wire than with silk thread, and on closing the knot the border of the skin is readily displaced, or there may be twists, that render the hold less secure;

this is especially apt to happen with our German wire, which has not yet attained the softness of the English. The pleasantest wires are those made of a mixture of gold and silver and of platinum, of which very fine, pliable, and, at the same time, firm wire may be made. [Very nice wire is made of lead, and it is supposed by some to be an advantage that this will break if the parts should swell excessively.] Still, how ridiculous it would be to try to substitute these expensive articles for ordinary silk, by which millions of wounds have been healed excellently, and will be in future! I pass over the many newly-recommended modes of fastening the wire by knots or twisting; they show that even those who advocate metallic sutures have found some trouble in fastening the knot. I first make a simple knot, draw it together, make two or three short twists, and cut off the ends close to the twisted part. Wire cuts the edges of the wound, just as silk does, if it be very fine.

I have rarely found the little objections to silk sutures sufficiently annoying to make me replace them by metal sutures. Beginners generally err in making sutures too tight; this constricts the edges of the wound. When they swell, as they mostly do, this constriction is rarely enough to kill the tissue at once, but causes inflammation with redness and suppuration about the puncture, which may spread and impede healing of the wound if the sutures be not removed in time.

Straight needles may be best introduced with the fingers; but curved needles, especially when they are small or the wound deeply seated, are introduced better and more certainly by means of a *needle-holder*. There are numbers of these; I am in the habit of using *Dieffenbach's*. It consists of a forceps with short, thick blades, between which we hold the needle firmly and securely, and introduce it through the skin in the direction of its curvature. This perfectly simple instrument suffices for almost all cases, and in good hands is surpassed by no instrument for security in holding and introducing the needle. Complicated instruments are especially suited for unskilful surgeons, says *Dieffenbach*, in the unparalleled introduction to his Operative Surgery; not the instrument, but the hand of the surgeon, should operate. Practice and habit render this or that instrument indispensable. Thus some find it complicated and inconvenient to seize the lips of the wound with forceps, as I taught you, although this is better than holding them with the fingers; for me, the latter would be very inconvenient. In this matter any one may do as his habits and inclination lead him. When I have to sew some deep part—as the velum, rectum, or vagina—I always use needles with handles.

Of course the number of sutures to be applied depends on the length of the wound; generally sutures half-an-inch apart suffice, but where perfect apposition and small cicatrices are very desirable, as in wounds of the face, they must be closer, and should alternate between coarse ones at a distance from the edge of the wound, and fine ones enclosing but a small portion of the edge.

The second variety of suture, *twisted* or *hare-lip suture*, is made by passing a long pin with a lance-shaped point through the flaps of the wound, and passing a strong cotton or silk thread around it, as I now show you. You take the thread in both hands, lay it parallel to and immediately over the pin, that is, transversely to the wound, pass it under the two ends of the pin from above, and draw on it, so as to approximate the edges of the wound exactly (this is the so-called *Nulltour*); now you change the threads to the other hands, and, with the right thread in the left hand, pass around the left end of the pin from above downward, and, with the left thread in the right hand, do the same for the right end of the pin; you change the threads again and make four to six similar, so-called figure of eight turns; then tie a double knot and cut the ends off close; then cut off the ends of the pin to a proper length, so that they may not press on the skin, but not so short as to prevent their being readily withdrawn subsequently.

There are a great number of other sutures, which for the most part are only of historical interest, and which we here pass over; some peculiar forms of suture will be treated in special surgery, under wounds of the different parts, as in wounds of the intestine.

Where are the advantages of the twisted over the interrupted suture? and when do you employ it? These indications may be reduced to two factors, so that you will consider the interrupted suture as the simpler and more common. The twisted suture is preferable—1. When the flaps of the wound are very tense; 2. When the skin-flaps to be united are very thin and without support—in short, where the lips of the wound have a tendency to roll in. The needle, remaining in position in both cases, renders the suture more secure and firm; the needle serves as a sort of subcutaneous splint for the edges of the skin; they are supported by it, and are also held more securely by the folds of thread on the outside. In many cases, in applying sutures in the face, the interrupted and twisted sutures are applied alternately; the latter serve as supports and to resist tension, the former to induce more exact union of the *edges of the wound.*

When the bleeding has been stopped and the wound united, all has been done that is at first necessary. Now let us observe what takes place in the closed wound.

Immediately after being united, the edges of the wound are generally white, from the pressure exercised by the sutures as they compress the capillaries; rarely the borders of the wound are dark blue; this always indicates great impediment to the return of blood through the veins, due to a loss of part of the blood-vessels. It is evident that the communication between arteries and veins may be greatly disturbed by the division of a large number of capillaries, so that at some point in the border of the wound the *vis a tergo* of the venous stream shall be insufficient. On the whole, this dark-blue color of the flaps of the wound is rare; it either disappears spontaneously or a small portion of the lip of the wound dies, a symptom to which we shall return when speaking of contused wounds, in which it is quite common.

Even after a few hours you find the borders of the wound slightly swollen and occasionally bright red; this redness and swelling are often absent (especially where the epidermis is thick), but occasionally, according to the extent and depth of the wound and tension of the skin, it spreads from two or three lines, or to as many inches, around the wound; the usual so-called local reaction about the wound takes place in this space. The wound pains slightly, especially on being touched. All this may be best seen in children and women with delicate skin. About wounds of the face, especially of the eyelids, we often notice extensive œdema in twenty-four hours; this frequently terrifies the friends, but is usually free from danger.

In a considerable number of cases, if the sutures be not too tightly applied, the edges of the wound appear unchanged not only at the time, but till the cure is complete. But often enough the wound shows the cardinal symptoms of inflammation; pain, redness, swelling, and increased heat, of which you may satisfy yourself by placing your finger on the parts about the wound, then on a distant part of the body. The process going on at the wound, and ending in the union of its edges, comes under the combination of morphological and chemical metamorphoses comprised by the name *inflammation*, and, in the case under consideration, would be termed *traumatic inflammation*, that is, an inflammation caused by an injury (τραυμα).

As a rule, these local symptoms have reached their height in twenty-four hours; if by that time they have not exceeded the above bounds, you consider the process as taking a normal course. *It is a marked peculiarity of traumatic inflammation, that, in a pure form, it is strictly limited to the borders of the wound, and does not extend without special cause.* It is not unusual for these symptoms to remain at the same height the second or even the third day; but by the third or fifth day, the redness, swelling, pain, and increased temperature, should have disappeared mostly or entirely. If the symptoms still increase the second, third, and fourth days, or if some of them, as severe pain, and great swelling, recur at this time, or if they remain at the same point to the fifth or sixth day, it is a sign that the course differs in some way from the normal. This will be especially evident from the general condition of the patient. The whole body reacts to an irritation of one part of it, not in a perceptible manner, in small wounds, it is true. We shall refer to this general reaction at the close of this chapter. At present, we shall consider exclusively the condition of the wounded part.

The third day, often indeed on the second, you may carefully remove the pins of the twisted suture, provided you have also applied interrupted sutures; this is best done by seizing the needle with *Dieffenbach's* needle-holder, and rotating it gently, while fixing the twisted threads with one finger. The threads usually remain as a sort of clamp on the wound, to which they are attached by dried blood; they subsequently loosen spontaneously; by forcibly detaching the thread, you would unnecessarily strain the wound, and possibly tear apart the freshly-united edges. If at this time we carefully feel the edges of the wound—if the œdema has subsided—we find them rather firmer than parts around; this state of *firm infiltration* sooner or later disappears.

When you have applied many stitches, you may remove some of them, that have little to hold, on the third day; others, on the fourth and fifth. At the tensely-stretched parts of the skin it is well to leave a few threads for eight days or more, or even leave them till they cut through the flaps of the wound, provided it can do any good to hold together the edges of the wound, which may be gaping open. Should the inflammation quickly exceed the normal amount, we must remove the sutures earlier, so that they may not increase the irritation; not unfrequently blood, that is decomposing or mixed with pus, at the bottom of the wound, is the cause of the unusual irritation.

In removing the interrupted suture, you should take the following precautions: cut the thread on one side of the knot, where you can most readily introduce the thin blade of the scissors without stretching the wound; then seize the thread at the knot with a dissecting forceps, and draw it out toward the side where it was divided, so as not to separate the edges of the wound by the traction.

Should you think that, after removing the suture, the union of the wound is still too weak to prevent its gaping, you may, by applying strips of ichthyocolla-plaster transversely over the wound, between the points where the sutures were, and fastening the ends (not the part over the wound) with collodion, give support enough to prevent tension of the flaps of the wound, such as unavoidably occurs in changes of expression in the face.

In from six to eight days, most simple incised wounds have adhered firmly enough to require no further support; indeed, in many cases, this is the case by the second or fourth day. If, in the course of the following days, the dry blood about the wound be carefully washed off, the young cicatrix appears as a fine red stripe, a scarcely visible fine line. This process of healing is called *healing by first intention.*

In the course of the subsequent months, the cicatrix loses its redness and hardness, and finally becomes perceptibly whiter than, and as soft as, the skin; so that for years it may be recognized as a fine white line. It often disappears almost entirely after some years. Some of you, who left the university with many still visible cicatrices on the face, may hope that they will be scarcely visible in six or eight years, when the Philistine visage will become you less than it does the student. Tempora mutantur et nos mutamur in illis.

## LECTURE V.

The more Minute Changes in Healing by the First Intention.—Dilatation of Vessels in the Vicinity of the Wound.—Fluxion.—Different Views regarding the Causes of Fluxion.

GENTLEMEN: You are now acquainted with the changes, visible to the naked eye, that take place in the wound while it is healing; let us now try to see what occurs in the tissues from the time of wounding till the formation of the cicatrix. For a long time, attempts have been made to study and know these changes more thoroughly, by making wounds in animals, and examining them at the different stages; but it is only the most exact microscopic examination of the tissue, and the direct observation of the changes after wounding, that have enabled us to give a description of the process of healing. I shall attempt to give you a brief *résumé* of the result of these investigations, which, until recently, I have made my special study.

[You will remember that I tried to explain to you the significance of traumatic inflammation as an accidental complication of the injury and of the healing of wounds. It is not long since this view began to gain ground; previous observers could not imagine wounds healing without inflammation, and their observations were made on tissues in a state of traumatic inflammation experimentally induced. Hence it is difficult to separate the phenomena due to healing of the wound from those forming part of the inflammation. At all events, observation of the healing of wounds first gave a clear insight into the nature of inflammation, and we may follow its course from this point of view.]

The interesting results thus arrived at have in a great measure brought it about that by "inflammation" we now mean generally the series of changes which we perceive on microscopic examination. Of late we are accustomed to consider these morphological processes as the essential part of the inflammation, and to term their occurrence and typical course the "inflammatory process." I would not weaken your interest in these things at the outset; but the prevailing tendencies render it necessary for me to call your attention to the fact that (as in all organic growth, and in each transformation of the tissues of the body) form is always the product of chemical or physical power inherent in the material supplied; the inflammatory, like every other physiological process, is chemico-physiological; this we never see, even with the best microscopes; we merely perceive the results of its action. These results, destruction and new formation of tissue, have something peculiar in their typical course; but they vary as widely as life and death; the tissue may die suddenly or not for years; of two neoplasia of the same structure, one may form in a few days, the other may require months; very different causes may induce very similar new formations. But I dread confusing you, if I enter further into the difficulties always arising when we speak of inflammation in general. So let me go at once into detail; and we will hereafter return to the general question of inflammation.

The changes after injury of the different tissues are particularly seen in the vessels, in the injured tissue itself, and in its nerves. The influence of the latter on the process is, however, so obscure, that we shall not consider it. We shall at once dismiss as unanswerable the question, whether the finest nutrient (vasomotor) nerves, which lose themselves in the different tissues (for the question can only arise concerning these), have any direct influence on the changes occurring in the tissues, and in the vessels themselves; and the rather so, as the ends of the nerves have only been certainly recognized in a few parts of the body, while for other parts it is entirely unknown how the nutrient nerves act, and what relation they have to the capillary vessels. You will have already had your attention called to the imaginable possibilities and probabilities on this point, in the lectures on physiology and general pathology. Hence, if we say but little about the nerves in what follows, it is because we know little of their action in this special process, not because we wish to deny their influence.

Let us first consider the simplest tissue; let us suppose a vertical section, through the connective tissue, with a closed capillary system at the surface of the skin, magnified 300–400 times. Here you have a diagram of such a system.

Let there be an incision down through the tissue; the capillaries bleed, the bleeding soon ceases, the wound is accurately united. Now what takes place?

The blood coagulates in the capillaries as far as the next branches, to the next points of intersection of the capillary net-work. Some coagulated blood usually remains also between the flaps of the wound;

FIG. 1.

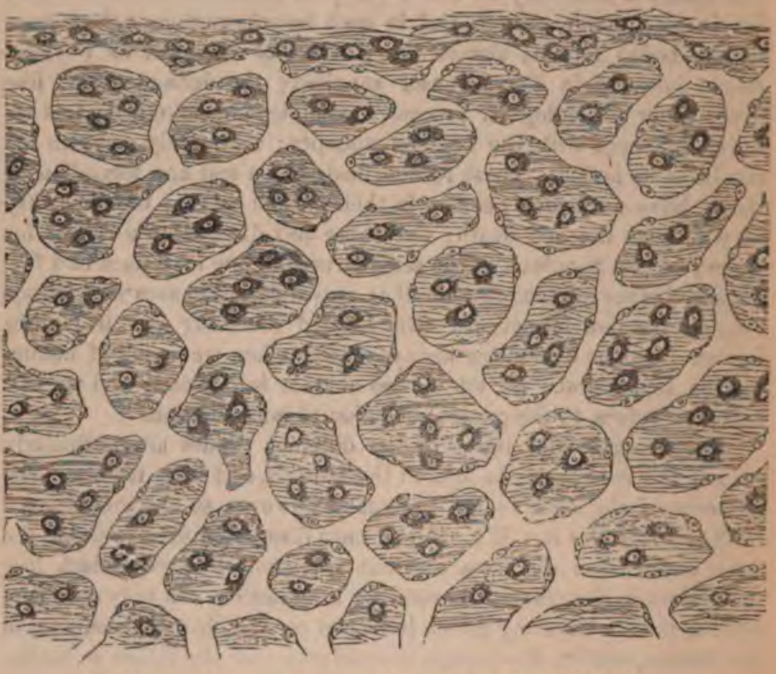


Diagram of connective tissue, with capillaries. Magnified 300–400.

we have omitted this in Fig. 2, so as to have the simplest possible representation of the changes. Of the channels for the circulation in our diagram, some have become impassable; the blood must accommodate itself to the existing by-paths—of course this takes place under a heavier arterial pressure than previously; this pressure is greater the greater the obstruction to the circulation, and the less numerous the by-paths (of the so-called *collateral circulation*). The result of this increased pressure is the distention of the vessels (which, however, is usually much greater than could be represented in the diagram), hence the redness about the wound, and to some extent also the swelling. But the latter also has another cause: the more the capillary walls are distended, the thinner they become; if under the ordinary pressure, with normal thickness of their walls, they permit blood plasma to pass to nourish the tissues, now under increased pressure, more plasma than normal will pass through the walls, which saturates

FIG. 2.

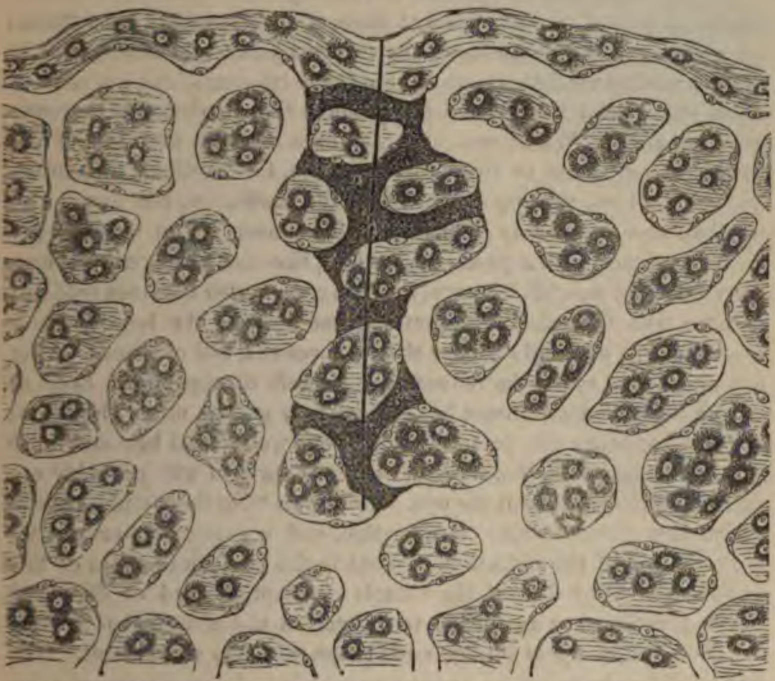


Diagram of incision.—Capillaries closed by blood-clot. Collateral distention. Magnified 300-400.

the injured tissue, and which the latter absorbs by its power of swelling.

This is a brief explanation of the perceptible changes in the borders of the wound, the redness and increased heat caused by the rapid development of the collateral circulation, by which more blood flows through the vessels nearer the surface; the swelling is caused by the distention of the vessels and swelling of the tissues, which again induces slight compression of the nerves, and this excites some pain.

This, as it seems to me, very simple mechanical explanation, would be much more valuable, if it fully explained the whole subsequent course, and could be applied to all inflammations, which are not of traumatic or mechanical origin. But this is not the case. Neither the great vascular distention that occurs some time after injury, that shows itself in extensive redness around the wound, nor the capillary dilatation that exists from the first in idiopathic inflammations, can be referred to purely mechanical causes.

If the disturbance of circulation through the incision be not extensive, it passes off very rapidly; these so-called *passive hyperæmias* are not exactly inflammations; their extent accurately corresponds to the mechanical conditions, while in regular inflammations the redness often extends far beyond the point where the circulation is mechanically impaired. We do not call it *inflammation* till irritation of the tissues accompanies, or in fact arises from, the capillary distention. Such irritations, causing dilatation of the capillaries, are numerous; we shall here speak only of the mechanical ones. You now see my ocular conjunctiva of a pure bluish white, like that of any normal eye. Now I rub my eye till it weeps, and the conjunctiva becomes reddish; perhaps with the naked eye you may see some of the larger vessels —with a lens you will also see the finer vessels, full of blood. After five minutes at most, the redness has entirely disappeared. Look at an eye where a small insect has accidentally gotten under the lid, as so often happens; the person rubs, the eye weeps and becomes quite red; if the insect be removed, in half an hour you will probably see nothing noticeable about the eye. Here you have the simplest observation how vessels dilate on irritation, and empty again soon after the cessation of the irritation. What is the immediate cause of this symptom? Why do not the vessels contract instead of dilating? These questions are as difficult to answer as the observation is easy to make, and to repeat innumerable times, with the same result. The fact itself has been known as long as man has observed; the old saying "ubi stimulus ibi affluxus" refers to this. The increased flow of blood is the answer of the vascular part to the irritation.

Of late, the process inducing this redness is called *active hyperæmia* or *active congestion.* *Virchow* took up the old name, and made "*fluxion* and *congestion*" again popular.

Assisted by your knowledge of general pathology, you will now perceive that it is desirable to give a theoretical explanation of symptoms which, through all time, have formed one of the most important objects of consideration in medicine, particularly as the process of inflammation is always considered as closely allied to this active congestion, or indeed even considered as always a sequent of the latter. *Astley Cooper*, a celebrated English surgeon, whose works you will read with pleasure, when you take up the study of monographs, a thoroughly practical surgeon, begins his lectures on surgery in the following words: "The subject of this evening's lecture is irritation; which, being the foundation of surgical science, you must carefully study, and clearly understand, before you can expect to know the principles of your profession, or be qualified to practise it creditably to yourselves, or with advantage to those who may place themselves under your care."

This will show you what part the questions to-day under consideration, which you might regard as a superfluous exercise of the mind and imagination, have played at various times; you will hereafter learn, from the history of medicine, that entire systems of medicine, of the greatest practical importance, are based on hypotheses that were formed for the explanation of this symptom in the vessels, of this irritability and of irritability of the tissues generally.

This is not the place to enter into a thorough historical consideration of this question; I will only call to mind a few hypotheses which have been advanced lately, under the already-existing knowledge of the vessels and parts visible to the naked eye, concerning the occurrence of vascular dilatation from irritation.

[The subject has become more comprehensible since *Exner's* recent work on the muscles which dilate the vessels, showing that there may be active dilatation of the vessels from nervous irritation, and that it is accomplished by contraction of the longitudinal fibres. If you imagine a hollow cylinder whose covering is of longitudinal muscular elements, and suppose these elements to contract, of course their transverse diameter increases, and there will be a corresponding increase of the calibre of the vessel which is made up of these elements. *Exner* has proved the truth of this explanation by simple experiment. We now seem certain that the circular muscles are contractors, the longitudinal dilators, of the vessels; and it is very possible that one set may be induced to contract independently, and that thus there may be an active dilatation. The opinion that the walls of the vessels, like the heart, are partly influenced by sympathetic, partly by cerebro-spinal nerves—the former inducing contraction, the latter regulating this contraction—seems gaining ground. Irritation of the sympathetic filaments would increase the contraction of the vessels, their division would cause paralysis of the circular muscles; such dilatation might also be induced by irritation of the cerebro-spinal nerves.

Having reached a point where you can regard dilatation of the vessels as an active reaction of the muscles of the vessels to nervous irritation, you will ask me whether experiment shows that the effect of the irritation can be actually observed in the living subject.]

Observers, who have of late persistently studied these questions, refer the continued capillary dilatation in acute inflammation to changes in the capillary walls, which are caused directly by the inflammation. *Cohnheim* claims that inflammation affects the walls of the vessels peculiarly, so that they are not only distended by the blood, but also become softer, of which more hereafter. *Samuel* ascribes inflammation to changes in the relations of the blood, walls of the vessels, and tissue, to each other. Up to the present we have not succeeded in finding accurately the chemical and physical changes in the walls of the vessels, which are known only by their results. This view is an advance beyond *Lotze's* view (according to which the molecules of the capillary walls separated from nervous irritation), inasmuch as no nerve-action seems to occur in this capillary dilatation developing in acute inflammation. This also corresponds with the demonstrations of *Schiff*, already mentioned, that the vascular dilatations forming after division of the sympathetic are not inflammatory, nor do they lead to inflammation without some further cause.

It is well known that, with the microscope, we can follow the circulation in the smaller arteries and veins, as well as in the capillaries of the web of the foot, in the mesentery and tongue of the frog, or in the wing of a bat; but the immediate effect of a mild chemical or mechanical irritant does not at once show in the capillaries, but first in contraction of the smaller arteries, occasionally also of the veins; this is very evanescent, of scarcely a second's duration, indeed, it often escapes observation, and we then suppose that its duration and grade are too slight for us to measure. This brief contraction is followed by the dilatation, whose immediate cause is indistinct even on microscopical observation. We shall soon see that the fluxion is the result of paralysis of the vessels.

*Virchow* appears to think that the irritation, which is certainly the immediate cause of the contraction, is followed by quick fatigue of the muscles of the vessels; that after a tetanic contraction there is a relaxation, just as in irritated nerves and muscles—a view which may find some support in a communication from *Dubois-Reymond* about the painful tetanus of the muscles of the vessels in the head as a cause of headache on one side, so-called hemicrania, since this supposed tetanus of the muscles of the vessels, induced by strong excitement of the cervical portion of the sympathetic, was certainly followed by their relaxation and great distention of the vessels, and shortly by symptoms of cerebral congestion.

But, in this view (by which a relaxation or temporary paralysis of the walls of the vessels and a consequent decrease of their resistance to the pressure of the blood would, it is true, be explained as a sequent of their contraction), we must not forget that it is by no means proved that the muscles of the vessels, once irritated and excited to rapid contraction, are indeed paralyzed, while in other muscles this fatigue usually occurs only after repeated irritation. It is necessary arbitrarily to assume that the muscles of the vessels very readily become fatigued, which is directly refuted by experiment. From physiology you know that *Claude Bernard* has proved that the contractions and dilatations of the arteries of the head are under the influence of the cervical portion of the sympathetic nerve, as I have already indicated. If we irritate the upper cervical ganglion of this nerve, the arteries contract; if we divide the nerve, there is dilatation (paralysis) of the arteries and capillaries. This experiment of irritating the muscles of the vessels may be often repeated, without their becoming quickly fatigued, unless the electrical current be too strong; hence we might imagine that there is little probability in the hypothesis of immediate fatigue after a single irritation. *Schiff*, however, like *Lotze*, assumes that active dilatation of the vessels is possible; he thinks that this necessarily follows from certain experiments; but this is perfectly incomprehensible to me, for there are no muscles that could actively dilate the vessels.

If the veins alone contracted on being irritated, filling of the capillaries would doubtless follow the obstruction, and there would then be no difference between venous (passive) hyperæmia and fluxion. But this supposition is quite untenable; it is perfectly incomprehensible that the veins alone should contract on inflammatory irritation. That the veins contract on mechanical irritation, you may see in the femoral vein of an amputated thigh, to which *Virchow* has called particular attention, and this irritability lasts even longer in the walls of the vein than in the nerves.

*Henle* already advanced the view that the symptom of distention of the vessels from irritation was directly caused by paralysis of their walls; when *Lotze*, in opposition to this, says that it is not supposable that there should be paralysis of the muscles in a man who is excessively irritated and has his muscles tense and his face glowing, his objection is not perfectly tenable. Nor does the other objection of the usually acute *Lotze* appear to me correct when he says, "What shall we think of paleness, of the contraction of the vessels that results from fright and terror? Does that look as if due to great muscular action, if redness in anger and shame is induced by paralysis?" I say this proves nothing. Fright may throw the muscles into a tetanic state, which is usually quickly followed by fatigue of the muscles of the vessels; immediately after a great fright, we generally feel the blood pour into the cheeks, as soon as we begin to breathe and recover from the shock; we soon grow red again, at first indeed redder than we often like; not unfrequently the paling from fright is often overlooked, and only the succeeding redness perceived.

Still, apart from these objections, how can we imagine the paralyzing action of an irritated nerve? We actually know such phenomena from physiology; the obstruction of the heart's action by irritation of the vagus nerve, of the movements of the intestines from irritation of the splanchnic nerve, etc. Here a vaso-motor nerve-system is supposed which arrests the contraction of the muscles; could not such a vaso-motor nerve-system also be supposed for the vessels—nerves, irritation of which lessens the tone of the muscles of the vessels and thus renders the walls less capable of resisting the pressure of blood? The doctrine about vaso-motor nerves is so difficult to explain, that even a brief exposition of the probable possibilities of the process would lead us too far; hence I must content myself with having called attention to the analogous physiological processes. *Virchow* and *Henle* agree in the view that the symptoms of fluxion are due to paralysis of the vessels, although they refer this paralysis to different causes; on the whole, most credence is attached to the view that the muscles of the vessels, like those of the heart, are partly under the influence of sympathetic, partly of cerebro-spinal nerves, and that the former cause the rhythmical (automatic) contractions of the vessels, and the latter act as regulators or obstructors of these contractions. Irritation of the sympathetic filaments would increase the contractions of the vessels, dividing them would result in paralysis of the muscles of the vessels and their consequent dilatation; but the latter might also be caused by irritation of the cerebro-spinal obstructive nerves.

The discovery by *Aeby*, *Eberth*, and *Auerbach*, that the blood-capillaries are entirely composed of cells, might excite new hypotheses about the irritability of the capillary cells and their influence on dilatation and contraction of the capillaries, although even this would not solve the mechanical difficulty which opposes the idea of an active vascular dilatation. In the action of local irritation and entirely local dilatation of the vessels we have the choice of considering that irritation of the nerves of the vessels (or of the living cell-substance of the capillary walls) directly disturbs their function, or that this disturbance is due to reflex irritation.

You have now material enough for meditation. None of the hypotheses advanced can claim to fully explain the symptoms of fluxion, although some of them perhaps contain the germ for future perfect development. Still the recognition of this truth, the distinction of hypotheses from observation, is useful; it does not limit the onward progress of experiment, but constantly reanimates it. Congratulate yourselves that it is permitted to you and the coming generation to clear up this point.

We shall now leave this question, and the next hour shall again return to the field of certain observation, to study the effect of the wounding on the tissue itself.

## LECTURE VI.

Changes in the Tissue during Healing by the First Intention.—Plastic Infiltration.—Inflammatory New Formation.—Retrogression to the Cicatrix.—Anatomical Evidences of Inflammation.—Conditions under which Healing by First Intention does not occur.—Union of Parts that have been completely separated

THE dilatation of the capillaries and the exudation of blood-serum that usually accompanies it, which we have found as the first effect of the wound, and which is most readily seen in the living tissue, as above mentioned, cannot of course by itself cause two flaps that are brought in apposition to unite organically—changes must take place on the surfaces of the wound, by which the latter are to a certain extent dissolved and melted into each other. For a long time union of wounds by first intention was represented as occurring just as you render two ends of sealing-wax soft by heat to fasten them together ; so here the substance itself was supposed to become the means of union. The union is really more like that by glue.

Let us keep in mind the above diagram (Fig. 2), and suppose that only connective tissue and vessels have been wounded, and that their reunion is the question for consideration. As you already know, connective tissue consists of cellular elements and filamentary intercellular substance. The cellular elements are partly the stable, *fixed*, long-known *connective-tissue corpuscles*, i. e., flat, nucleated cells, with long processes, which adhere to the connective-tissue bundles, partly the *wandering cells* discovered by *Recklinghausen*, which are identical with white-blood and lymph cells, in form, species, and vital peculiarities, are probably formed for the most part in the lymphatic glands, through the lymphatics enter the blood, from the capillaries and veins, occasionally wander into the surrounding tissue (as discovered by *Stricker*), there become fixed tissue-cells, or again (as observed by *Hering*) enter the lymphatic or blood vessels, or undergo metamorphoses not yet discovered.

If we examine the tissue of the flaps of the wound a few hours after the injury, we shall find it full of wandering cells. These increase enormously from hour to hour; they infiltrate the fibrous tissue, already softened by swelling, and even wander from one flap of the wound to the other. During this cell-activity, and probably on account of it, the connective-tissue intercellular substance gradually changes to a homogeneous gelatinous substance, which gradually disappears as the cells increase, possibly being consumed by them ; so that there is a time when the surfaces of the wound in apposition consist almost entirely of cells, held together by a very slight quantity of gelatinous intermediate substance (which subsequently becomes firmer and finally fibrous).

In the sketch below (Fig. 3), a sequel to the above diagram, you

FIG. 3.

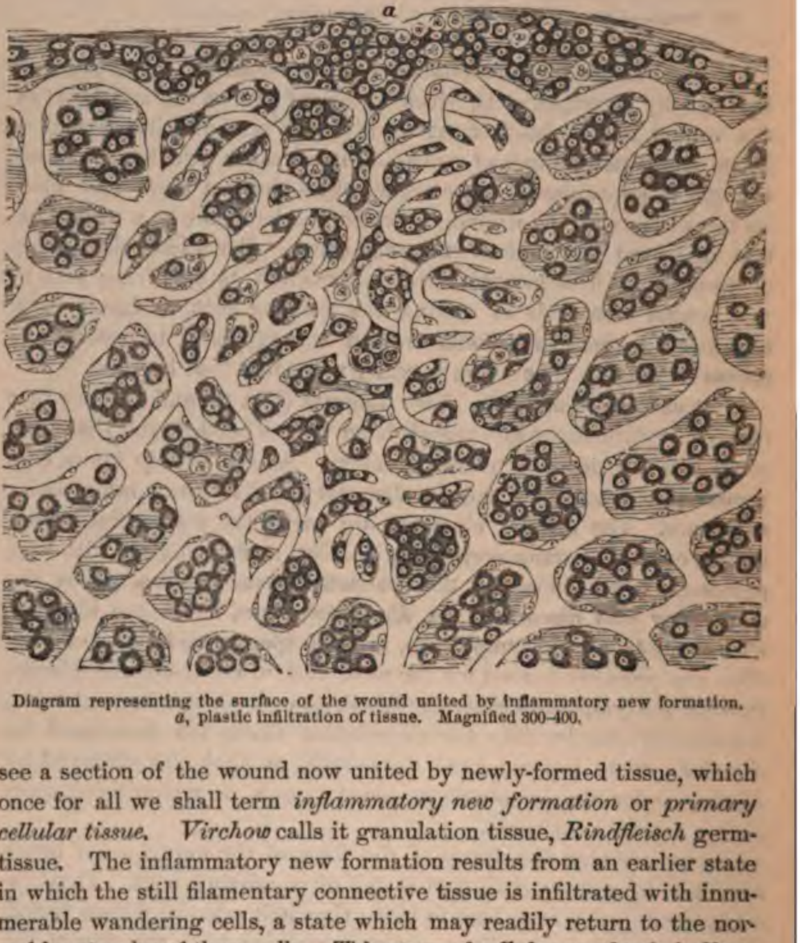
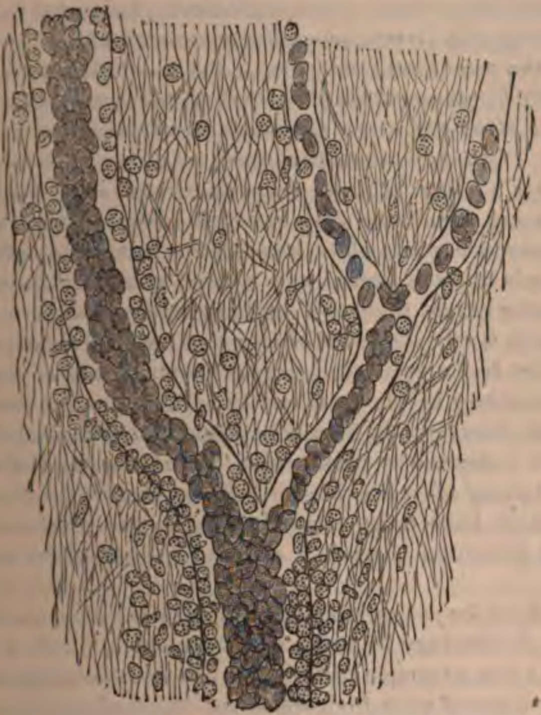


Diagram representing the surface of the wound united by inflammatory new formation. *a*, plastic infiltration of tissue. Magnified 300-400.

see a section of the wound now united by newly-formed tissue, which once for all we shall term *inflammatory new formation* or *primary cellular tissue.* *Virchow* calls it granulation tissue, *Rindfleisch* germ-tissue. The inflammatory new formation results from an earlier state in which the still filamentary connective tissue is infiltrated with innumerable wandering cells, a state which may readily return to the normal by atrophy of these cells. This stage of *cellular* or *plastic infiltration*, in which the tissue feels firmer than in watery *edematous infiltration*, is almost always at some distance from the edge of the wound, so that in any such specimen of a recent wound we may follow the development of the inflammatory new formation from the plastic (cellular) infiltration, if we make microscopical examinations from the normal tissue toward the wound. The injury represents an inflammatory irritation, whose action may extend somewhat beyond the immediate vicinity of the irritation, but then rapidly diminishes.

FIG. 3 A.



Vein and capillary vessel from the mesentery of a frog, which has lain exposed for some hours. Red blood-cells from the circulation; white blood-cells lying against the walls and wandering into the loose connective tissue of the mesentery. Magnified about 800 diameters.

In the great majority of cases there will be at least a slight layer of coagulated blood between the flaps of the wound; this also extends somewhat into the interstices of the tissue of the flaps of the wound. This blood-clot may sometimes interfere with the healing, as when, from its size or other casues, it decomposes or turns to pus; but it may also become cicatricial tissue and perfectly disappear with the new formation in the flaps of the wound; this must take place for union by the first intention to occur. We shall hereafter speak of the changes that take place in the clotted blood during this process.

We must now attend to the question, Whence come the innumerable wandering cells that infiltrate all inflamed tissues immediately after their irritation, as they here do the flaps of the wound? Of late, this question has received the following wonderful explanation, which ten years ago would have been considered as the fancy of a madman. *Cohnheim* made the following remarkable observation: he introduced finely-powdered anilin blue into the lymph-sac in the back of a frog, then irritated the animal's cornea with caustic, and found that numbers of wandering cells (lymph-pus cells) containing anilin gradually collected at the cauterized point; hence the conclusion, *at an irritated point white blood-corpuscles wander from the vessels into the tissue; these white blood-corpuscles constitute the inflammatory cellular infiltration.* *Cohnheim* then confirmed, by direct observation on the mesentery of a living frog, the discovery already made by *Stricker* on the nictitating membrane that had just been removed, that under some circumstances the white blood-cells wander through the walls of the vessels into the tissues, and showed also that this occurred to a still greater extent in dilated capillaries and veins.

Although it was afterward shown that an English experimenter, *Aug. Waller*, had several years previously made similar observations on the mesentery of the toad and the frog's tongue, the works of the German observers, *Stricker*, *Von Recklinghausen*, and *Cohnheim*, were quite independent of his, and *Cohnheim* has the undivided honor of having correctly interpreted his observations on inflammation, which have constantly advanced to the present time, and of having presented them in a form to greatly affect all modern pathology.

It is difficult for you, gentlemen, to imagine the immense impression made on all histology by these new discoveries, which I have just imparted to you as simple facts, because you are not acquainted with the former point of view from which the origin of inflammatory new formations, and that of complicated organized growths, was regarded. From previous observation, our idea of the affair was about as follows: It was supposed that the cells of the connective tissue, of which only one variety, the fixed, was known, increased greatly by division as a result of irritation, and cellular infiltration thus resulted. Imagine yourselves back a few years, in a time when nothing was known of the vital peculiarities of young cells, of their amœboid and locomotor action, and we only knew how to deduce the course of the pathological process from various stages of the diseased but not dead tissues, as is still the case in the normally-developing layer; then you will readily understand that it was decided without hesitation that the cells lying packed together in the inflamed tissue were formed out of one another. Even this was a great advance, which was only possible after the overthrow of the *generatio æquivoca*; for, not long before, the development of cells and tissue from lymph, coagulated blood, and fibrine, was firmly believed in. The first observations on cell-division as a result of abnormal irritation were made on cartilage by *Redfern* in England; then followed the observations of *Virchow* and *Heis* on inflamed cornea. In both cases it was seen that after cauterization with nitrate of silver, or after introduction of a seton, the tissue was filled with young cells; in the original tissue-cells, biscuit-shaped, then double nuclei were seen, from which a division was decided on; young cells were seen grouped together, and their origin from the tissue-cells seemed indubitable. Hence arose the idea that inflammation was a process in the tissues, which, entirely independent of the vessels, was associated with a rapid luxuriant proliferation of tissue-cells, and partial softening and disintegration of the intercellular tissue. *Von Recklinghausen's* discovery of the two varieties of cells found in connective tissue, as well as his discovery of the varied movements of pus-cells, might well have given rise to the question whether the proliferation of the cells, on irritating the tissue, started from the fixed or movable connective-tissue corpuscles, but failed to do so. But now observation is piled on observation; and we are driven to the supposition *that all young cells which in inflammation we find abnormally in the tissue are wandering white blood-cells.*

Observers who have recently investigated this point do not all agree; some still ascribe to the stabile cells of the connective tissue a part in the inflammatory process. *Stricker*, in his latest publications, maintains that, on irritation, the stabile tissue-cells are filled with neoplasma, increase by segmentation, and aid in the formation of pus; but he does not deny the wandering of white blood-cells. *Cohnheim*, *Key*, *Eberth*, and others have denied the correctness of *Stricker's* observations, or rather of his interpretations. Observations on this point are so tedious that we cannot wonder at the delay in elucidating a question apparently so simple.

Of course, from the various errors to which we are liable in interpreting the significance of what has been observed, we should be very careful about announcing general principles. In regard to the inflammatory changes in connective tissue, however, as far as my observation and criticism extend, I would maintain the above statements.

In cartilage alone, nothing has been observed different from former appearances. As the hyaline cartilage-substance has no canals passable for cells, so far as we at present know, there is little left except to suppose that the increase of cells in the cartilage-cavities after irritation results from division of the protoplasm of the cartilage-cells; of this I shall hereafter show you preparations; still hyaline cartilage has never yet been watched for days in a living and irritated state, and consequently this observation must give place to the studies on living connective tissue.

But in hyaline cartilage there is no such acute suppuration or infiltration of pus as in connective tissue. I will again repeat that I only consider a renovation and proliferation of connective tissue and corneal cells as improbable in those cases where the protoplasm has been entirely metamorphosed even to the nucleus, that is, the stabile connective tissue and corneal granules of grown animals whose tissues resemble those of man. It has never been doubted that protoplasm, when it exists as such in cells, that is, in growing tissues of young individuals, may increase and divide up; inattention to these points may have given rise to some of the differences in the views above stated. The same is true of epithelial tissues; it has never been maintained that the cells of fully-developed epithelial tissue, the elements of the hair, nails, epidermis, and upper layer of flat epithelium, could be renovated by irritation, while it is not denied that constant increase of the young elements of these tissues is a physiological necessity for their growth; here the only difference is that growth of these epithelial tissues continues during life, while that of connective tissues only goes on to a certain age, and hence, after cessation of the growth, wandering cells are the only young elements found in these tissues.

If it be now established beyond doubt that most of the young cells which infiltrate the inflamed tissue and sometimes escape from it as pus, as we shall hereafter see, are white blood-cells, or briefly *wandering cells*, then two questions arise: Why do so many cells wander into inflamed tissue? How do such numbers of them get into the blood, and whence do they come? There are different views as to the mode of escape of wandering cells through the walls of vessels.

My views are as follows: The first change that we see in inflamed tissues during life is dilatation of the vessels; the immediate result of this is increased transudation and a collection of white blood-cells along the periphery of the vessel. Then the wall of the vessel is gradually softened by some unexplained chemical process that goes on in every inflammation, so that by their active movements the white blood-cells gradually enter and finally pass through it. Hence dilatation of the vessels, accumulation of white cells along the walls of the vessels, and softening of the walls, seem to me to be the requirements for extensive emigration of cells. *Cohnheim* and *Samuel* have lately announced the same opinion. Whence come the immense number of white blood-cells that escape in inflammation is entirely a physiological question. The lymphatic glands and the spleen are the organs which we first suspect; and, although it cannot be proved that numerous new lymph-cells are formed as the others escape, it is very probable, especially as we know from clinical experience that the lymphatic glands in the vicinity of an inflammation almost always swell. In spite of careful search, I have been unable to discover any thing about the morphological process of this cell-formation, but consider it very probable that lymph-cells originate from sprouting of the nets of the lymph-sinuses in the glands.

I must mention one other point, which is, that in inflammation red blood-corpuscles also not unfrequently pass through the walls of the vessels; according to *Cohnheim's* experiments, this is greatly influenced by the increased intravascular pressure.

According to *Arnold*, not only red but white blood-cells escape from the walls of the vessels at points where the capillary vessels leave small openings (stigmata, stomata); it is said to be more especially the cement of the cells of the capillary vessels that swells on inflaming, and becomes so yielding that fine streams of blood-serum flow through these vessels into the interstices of the tissues.

Let us now return to our wound, and see what becomes of the tissue infiltrated with cells, of the inflammatory new formation; how the cicatrix develops from it while the cell-infiltration extends slowly and sluggishly at some distance from the wound. The cells in the surfaces of the wound, which already adhere loosely, gradually assume a spindle shape; the intercellular tissue then becomes firmer, the spindle-cells change to fixed connective-tissue cells, and finally the young cicatricial tissue assumes more and more the form of normal, fibrous connective tissue; that is, the white blood-cells become fixed connective-tissue cells, as probably takes place even in the embryo. Here, again, we are met by various questions. The newly-formed adhesive, interlacing tissue soon becomes firm, especially in healing by the first intention; even after twenty-four hours we find its intercellular substance quite stiff and fibrinous, and the borders of the wound are also more or less infiltrated with this stiff substance; it is only the early hardening of the intercellular connective substance, formed of transuded serum and softened connective tissue, that explains why the union is so firm, even the third day, that the flaps of the wound hold together without sutures, for without such connective substance the young cellular tissue could not be so coherent. This stiffening connective-tissue substance (Fig. 8) is most probably fibrine, which consists of the transudation coming from the vessels under the influence of the extravasated blood-corpuscles, possibly also of the wandering cells. From the excellent experiments of *Alexander Schmidt* it is known that most exudations contain the so-called fibrogenous substance, which forms fibrine as we know it in the coagulated state, by combining with the fibro-plastic substance of the blood and other tissues. Very accurate proportions of fibrogenous and fibrino-plastic substance are required to form fibrine; these favorable requirements occur in many inflammations. *Schmidt* considers it probable that all firm fibrous tissue is formed and maintained by the fibrogenous substance from the blood being precipitated in a certain manner around the tissue-cells, because they contain a fibrino-plastic substance in a firm shape. Under this hypothesis we must suppose a specific cell-action, which would cause the coagulating product to assume the form of muscular striæ in one place and in another of connective tissue. In our case this is a very probable view, for we see filamentary connective tissue gradually form from the intercellular coagulated fibrine. It is true the amount of intercellular substance in the new formation is not great, but there is little doubt that the small spaces between the cells are filled by it. A short time subsequently the young cicatricial tissue appears still to consist chiefly of spindle-cells closely pressed together (Fig. 9); but then the spindle-cells diminish greatly by flattening, many are even destroyed, and we have now a filamentary connective-tissue substance, which is to be considered partly as a product of secretion, partly as metamorphosed protoplasm of the spindle-cells; the cicatricial tissue finally remains stable in this state. *Thiersch*, who quite recently has again carefully studied the healing of wounds, maintains that the apparently fibrinous intermediate substance is not fibrine, but only metamorphosed connective tissue.

I will not deny that there may be an immediate union of the softened edges of the wound, although it must be very rare. Quite recently I had Dr. *Gussenbauer* make a new series of accurate observations on healing by the first intention with especial reference to *Thiersch's* views. He could not confirm the latter's observations, but he, as well as *Güterbock*, arrived at results which in the main correspond with the above views, which I arrived at from my own studies.

[Investigations of *F. Fuchs* render it probable that in the cornea, within a few hours after cauterization, there is active wandering of colorless blood-cells, while later the corneal corpuscles proliferate and bring about detachment of the slough as well as replace the destroyed tissue.

*Felix v. Winiwarter* has shown that normally the spaces between the cells of the capillary walls are permeable to injections under the ordinary pressure of the heart's action, consequently, for the blood-plasma; but that into inflamed tissue the injection passes in much greater quantity and at more places under the same pressure.

The cellular new formation is probably composed of the wandering cells and the tissue-cells which are capable of proliferation. It has long been known that cells whose protoplasm has all been changed and which no longer show any vital changes will, under certain irritations, rejuvenate and act as embryonal cells rich in protoplasm. But experiments of *Ziegler* and *Tillman* show that the inflammatory new formation may develop from wandering cells alone. *Ziegler* cemented two covering glasses in such a way as to leave a space between them into which fluid and wandering cells could enter; these he placed in the abdomen of living animals, leaving them there for a month. Then, after treating with osmic acid they were placed under the microscope, and wandering cells found between the glasses developed to a cellular tissue. To completely exclude the proliferation of normal tissue-cells, *Tillman* placed sections of tissue, hardened in alcohol, with incisions as in a wound, in the abdomens of living animals, and the incision became entirely closed by wandering cells, vessels were formed, and the wandering cells transformed to connective tissue.

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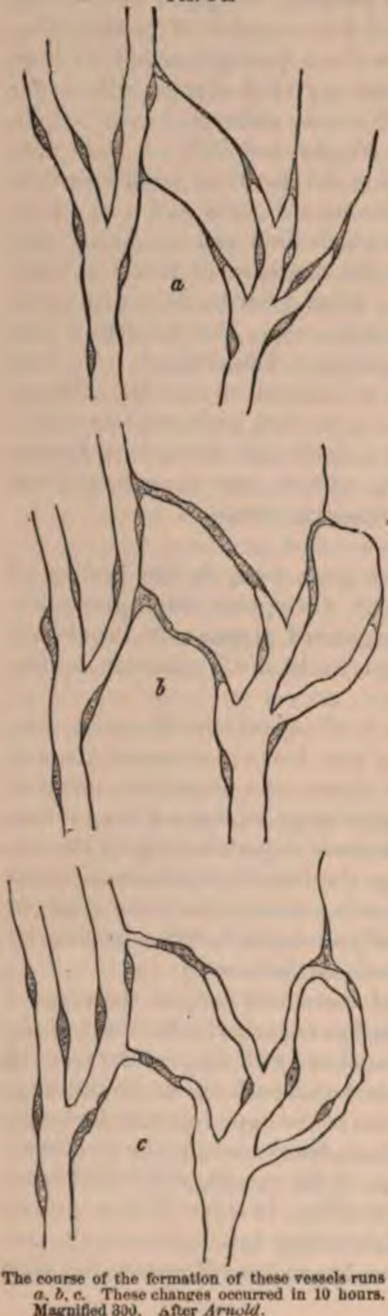
To recapitulate what we have gone over, in the healing of wounds by first intention, we may distinguish three grades: 1. Union by an organic cement, coagulated plasma; 2. Provisional union by a cellular neoplasia; and, lastly, 3. Cicatrization by filamentary connective tissue.

The healing process is the same in all organs—in the cornea, skin, muscles, nerves, even in cartilage and bones; of course, there is some variation dependent on the tissue. An *immediate* union of the divided parts, as pictured by some surgeons, has not been shown by accurate examination; wounds made experimentally in the cornea with the sharpest knife always show an inter-substance, which may, it is true, be microscopic, but is a scar in the strict sense of the word. Hence it is theoretically incorrect to term healing by first intention as a complete *restitutio ad integrum.*]

Meantime, what has become of the closed ends of the vessels? The blood-clot in them is reabsorbed or organized; the walls of the vessels send out shoots which communicate with the vascular loops of the opposing border of the wound, and with each other. In this way, however, only the rather scanty union of the opposing vascular loops, which is at first slight, is accomplished; these were already formed by extensive tortuosities and windings of the vessels, which had loop-shaped terminations after the injury (Figs. 12–14). This is not the place to go into the details of this interesting development of the vascular loops; their development is not due solely to dilatation, but very

FIG. 3 B.



The course of the formation of these vessels runs *a*, *b*, *c*. These changes occurred in 10 hours. Magnified 300. After *Arnold*.

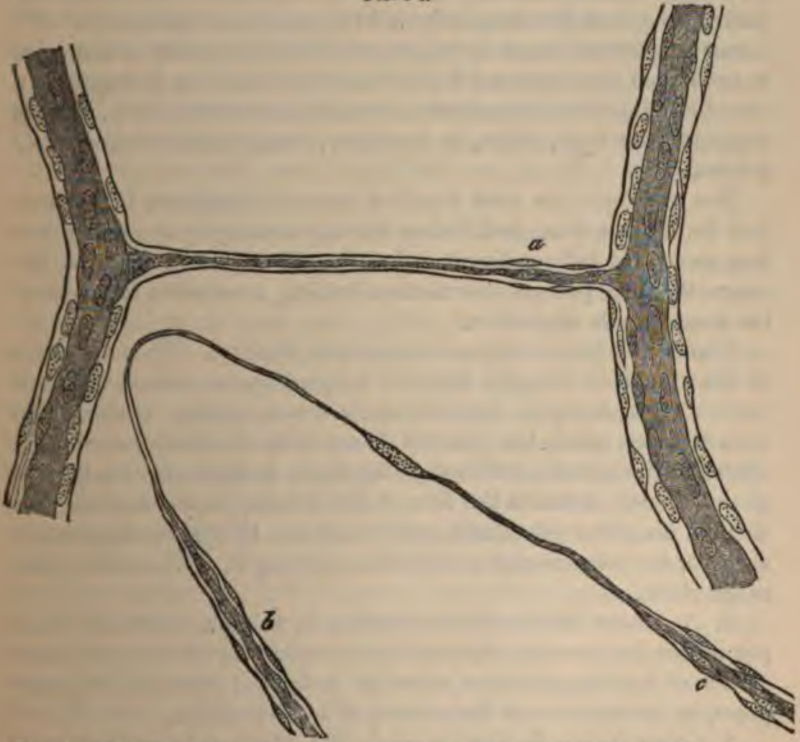
much to interstitial growth of the walls of the vessels. The original, formerly-existing vascular union is thus replaced by a newly-formed vascular net-work which is at first far richer.

Quite recently *Arnold* has most carefully studied the process of the development of vessels, and has seen it go on in the tails of tadpoles (Fig. 3 B.)

Although the heart and larger vessels of the embryo seem to originate from appointed cell-groups of the middle germinal layer, by the peripheral parts forming the wall of the vessel and the central parts the blood-cells, later this does not seem to occur; at least, observations made on this point by *Rokitansky* and others do not seem to have obtained much credence. According to *Arnold's* investigations, offshoots from the vessels seem to be the only mode of development of vessels in the embryo.

I formerly thought that there must be another mode of growth for vessels in the formation of granulations and in some neoplasia, namely, a tubular formation by laying together spindle-cells, as at *a*, *b*, and *c*, in Fig. 3 C; this I called "secondary" formation of vessels ("primary" I applied to the development of the heart and larger vessels in the middle germ-layer). The development of vessels by offshoots I called "tertiary." But, since recent investigations, I readily agree that the mode I termed "secondary" possibly did not exist, and that the fine plasma string (the offshoot) and the fine tube, on which the spindle-cells growing out of the young adventitia lay, may have escaped my notice. But I will not neglect to mention that *Thiersch*, supported by recent observations, has repeated his former assertions (which then seemed to me improbable) that in the young inflammatory neoplasia there is a net-work of tubes, connected with the blood-vessels by stigmata, which is bounded merely by the tissue-cells, not by special walls ; this agrees very well with the recent observations on capillaries in inflamed tissues. According to this, there would be blood-vessels in this tissue which are not circular canals, but irregular intercellular passages, perhaps merely bounded by spindle-cells.

FIG. 3 c.



Disposition of vessels in the vitreous body of an embryo calf. Magnified about 600. After *Arnold*.

As a result of the restoration of circulation through the young cicatrix, the circulatory disturbances caused by the injury are removed, the redness and swelling of the borders of the wound disappear ; from the numerous vessels, the cicatrix appears as a fine red stripe. Now the consolidation of the cicatrix must take place: this is accomplished, on the one hand, by the partial disappearance of the newly-formed vessels, whose walls fall together, and they thus become solid, fine, connective-tissue strings; on the other hand, by the intercellular substance becoming firmer and containing less water, as above mentioned, the cells assume the flat form of connective-tissue corpuscles, or disappear; possibly some of them remain as wandering cells, and return again into the lymphatics or blood-vessels. To this condensation and atrophy is due the great contractile power of the cicatricial tissue, by means of which large, broad cicatrices may occasionally be reduced to half their original size.

At the first glance, it might appear to you contradictory, that an apparently excessive capillary net-work should be formed in the young cicatrix, and should subsequently be for the most part obliterated. We cannot explain this apparent excess, still there are plenty of analogies in embryonal development; I only need to remind you that there is a period in fœtal development when, even in the vitreous body, there is a capillary net-work, which, as you know, disappears, leaving *scarcely a trace.*

Not to fatigue you with so-called theoretical subjects, I leave this field for a short time, and, before leaving healing by the first intention, as a point fully understood, I shall make a few remarks on the causes that may prevent this mode of healing, even when the flaps of the wound are in apposition.

Healing by first intention does not take place: 1. When the edges of the wound are brought together by plasters, or sutures, but their tension or tendency to separate again is very great. Under these circumstances, either the plasters do not keep the wound accurately closed, or the sutures cut through the flaps; perhaps also the tension of the tissues obstructs the flow of blood in the capillaries, and thus disturbs the cell development and formation. How great this tension must be, and what means we have for relieving it, you can only learn in the clinic.

2. A further obstruction to healing is, a large amount of blood poured out between the edges of the wound; this interferes with the process of healing, partly as a foreign body, and partly, if it decomposes, by the influence of the process of decomposition.

3. Other foreign bodies, as sand, dirt, alkaline urine, fæces, etc., also retard the healing, partly mechanically, partly chemically. Hence these substances should be carefully removed before uniting the wound. In wounds of the urinary bladder, it is not usual to attempt the closure of the skin-wound; the urine would force its way into the subcutaneous cellular tissue, or into the peritoneal sac, and excite terrible

injury. Here, under some circumstances, it would be a decided fault to unite the wound, although of late the views on this particular point differ somewhat from those of former days.

4. Lastly, from a contusion, whose effect on the flaps of the wound we may fail to observe, there may have been an extensive disturbance of circulation and destruction of minute tissue, which has induced the partial death of certain parts or of the whole surface of the wound. Then, as there is no cell-formation in the edges of the wound, but only where the tissue is still living, we have small tags of the destroyed tissue lying as foreign bodies between the edges of the wound; these must prevent healing by first intention. If this mortification attack only minute particles, these may possibly quickly undergo molecular disintegration and absorption; this may occur not unfrequently. We shall speak more extensively of this mortification of the tissue, and of its detachment from the healthy parts, when treating of contusions.

Experience, arising from many observations in judging of wounded surfaces, will hereafter enable you in most cases to say whether healing by first intention may be expected or not, and you will also learn when it may be useful, even in doubtful cases, to try to aid this union by applying dressings.

You will occasionally hear of wonderful cases where parts of the body, completely separated, have again become united. This appears to be actually the case. I have never had the opportunity of making any observations on such cases; still, even in late days, very trustworthy men have asserted that they have seen small portions of skin again unite after being removed from the fingers by a blow or cut, then carefully replaced and fastened on with adhesive plaster. Formerly I contended against the possibility of this healing, but must now admit it, also on theoretical grounds, after it has become imaginable that, through the movements of the cells, the detached portions, if not too great, may soon be restored to life again by the entrance of wandering cells. That we may successfully transplant a twig, cut from one tree, into another one, is well known; but, as the circulation in plants is not by pumping, but the sap runs simply by cellular force, the analogy is not very close; it was more remarkable, it is true, that a cock's spurs could be transplanted to his comb, but between birds and men the differences in the formative process are also very great, and any immediate transfer of observations is inadmissible in practice.

When treating of the cicatrization of wounds with loss of substance, we shall investigate the discovery of *Reverdin* that we may cause epidermis to grow on granulating surfaces.

[Before the formation of vessels, blood-plasma may circulate in the tissue, which is very important for its nutrition. This *plas-*

*matic circulation*, immediately after the injury, from dilatation of the capillaries, may be very important for the edges of the wound itself, their vessels having been thrombosed. Even normally, to some extent, streams of blood-plasma may escape through the walls of vessels and spread around in the tissue.

Portions of the body that have been separated may directly adhere, when reapplied, if the conditions are good for this plasmatic circulation. The divided part here acts as young cicatricial tissue previous to its vascularization ; it must first be nourished if organic union is to result. The size is the most important factor ; a small piece with large surface is more readily saturated with plasma. The structure is also important ; it is an advantage if the tissue contains numerous channels for the plasmatic circulation ; fatty tissue is the most unfavorable for this, as the channels are occupied by fat-cells. When the separated part is once nourished by the plasma, the union goes on well ; white blood-cells wander into the divided portion, and the whole is nourished by plasma till the neoplastic vessels grow into the divided part, and normal circulation is restored.

The union of pieces of skin to granulating surfaces would go on in much the same way. These pieces may be introduced some hours after death of the part from which they are taken. Dr. *E. P. Brewer* (*Medical Record*, May, 1882) has made some experiments which seem to place thirty-six hours as the limit of this time.

A common instance of readherence of divided parts is the reimplantation of teeth that have been pulled by mistake.]

In his history of plastic operations *Zeis* has collected all published cases of reunion of parts that had been entirely separated. *Rosenberg* has completed this list to the present time, and gives a number of cases carefully observed by himself where portions of noses and fingers which had been cut off reunited. He confirmed previous observations that the epidermis and occasionally small portions of the surface of such parts became gangrenous, while healing went on below.

## LECTURE VII.

Changes perceptible to the Naked Eye in Wounds with Loss of Substance.—Finer Processes in Healing with Granulation and Suppuration.—Pus.—Cicatrization.—Observations on "Inflammation."—Demonstration of Preparations illustrative of the Healing of Wounds.

It now remains for us to inquire what becomes of the wound, if, under the above circumstances, it does not heal by first intention. Then, as the flaps gape, we have an open wound before us; and the conditions are the same as if the gaping wound had not been closed, or as if a piece had been cut out, as in a wound with loss of substance. Accurate observation of such wounds, which are usually covered with some unirritating body, as with a fold of linen dipped in oil, with oiled or dry charpie, etc., shows the following changes—if we examine it daily, this is not necessary it is true, and may even be injurious: after twenty-four hours, you find the borders of the wound slightly reddened, somewhat swollen, and sensitive to the touch; the same symptoms as in closed wounds. As in healing by first intention, these symptoms may be very insignificant or entirely absent, as in old, relaxed, flabby skin, also in strong skin with thick epidermis. We observe these symptoms best in the skin of healthy children. An extensive and increasing redness, swelling, and pain about the wound, make us suspect an abnormal course; just as, with the same symptoms in a wound healing by first intention, various individual circumstances are to be considered, and the vibrations from the normal to the abnormal are so numerous, that the dividing line is often difficult to determine. After the first twenty-four hours, the surface of the wound has changed but little; all over it you can still recognize the tissues quite distinctly, although they have a peculiar gelatinous, grayish appearance; you also find a considerable number of yellowish or grayish-red small particles over the surface; on close examination, you find these to be small fragments of dead tissue, which still adhere, however. The second day, you may already notice a trace of reddish-yellow, thin fluid over the wound, the tissues appear more regularly grayish red and gelatinous, and their boundaries become more indistinct. The third day, the secretion from the wound is pure yellow, somewhat thicker, most of the yellow dead particles are detached and flow off with the secretion; the surface of the wound becomes more even and regularly red—it *cleans off*, as we say technically. If you had not bound up the wound (a stump from amputation, for instance), and had received in a basin the secretion that formed, the first and second day you would find it bloody, brownish red, then of a gelatinous dirty gray, then dirty yellow: at the points where the secretion flows from the wound, fibrine not unfrequently stiffens in drops. If you examine carefully with a lens, even the third day, you will see numerous red nodules, scarcely as large as a millet-seed, projecting from the tissue —small granules, *granulations*, *fleshy warts*. By the fourth or sixth day these have greatly developed, and gradually join into a fine, granular, bright-red surface—the *granulating surface;* at the same time, the fluid flowing from this surface becomes thicker, pure yellow, and of creamy consistence; this fluid is *pus*, and, when of the quality here described, it is good pus, *pus bonum et laudabile* of old authors.

Of this normal course there are many varieties, which chiefly depend on the parts injured, and the mode of injury; if large shreds of tissue from the surface of the wound die, the wound is longer in cleaning off, and then you may sometimes see the white, adherent shreds of dead tissues still clinging for days to the surface, most of which is already granulating. Tendons and fasciæ are particularly apt to have their circulation so impaired, even by simple incised wounds, that they die to an unexpected extent from the cut surface, while there is little loss of loose cellular tissue or muscle. This is undoubtedly due partly to deficient vascularity of the tendinous parts, partly to their firmness, which does not permit rapid collateral dilatation of the vessels; the same is true in injuries of bone, especially of the cortical substance, where there is often death of the injured bone-surface, that requires a long time for detachment. Other obstacles to active development of granulations are constitutional conditions; for instance, in very old or debilitated persons, or badly-nourished children, the development of granulations will not only be very slow, but they will look very pale and flabby. Hereafter, at the close of this chapter, I will give you a short review of those anomalies of granulation which are daily occurrences in large wounds, and, to a certain extent, may be regarded as normal or at least customary.

But, to return to the observation of the normally-developing layer of granulations, with the continued secretion of pus, you perceive that the granulations become more and more elevated, and sooner or later attain the level of the skin, and not unfrequently rise above it. With this process of growth, the individual granules become thicker, and more confluent, so that they can hardly be recognized as separate nodules; but the entire surface assumes a glassy, gelatinous appearance. Occasionally the granulations remain for a long time at this stage, so that we have to use various remedies to restrain the proliferating neoplasm within bounds that are requisite for recovery; on the periphery, particularly, the granulations should not rise above the level of the skin, for the cicatrization has to commence at this point.

The following metamorphoses now gradually occur: the entire surface contracts more and more, becomes smaller; on the border, between skin and granulations, the secretion of pus diminishes; first, a dry, red border, about half a line broad, forms and advances toward the centre of the wound, and, as it progresses and traverses the granular surface, it is followed closely by a bluish-white border, which passes into normal epidermis. These two seams result from the development of epidermis, which advances from the periphery toward the centre; *cicatrization* begins; the young cicatricial border advances half a line or a line daily; finally, it covers the entire granulation surface. The young cicatrix then looks quite red, and is thus sharply defined from the healthy skin; it feels firm, more so than the cutis, and is still very intimately connected with the subjacent parts. In the course of some months, it gradually grows paler, softer, more movable, and finally white; in the course of months and years, it grows still smaller, but often remains whiter than the cutis all through life. The strong contraction in the cicatrix often causes traction on the neighboring parts, an effect that is occasionally desirable, but sometimes very unwelcome, as, for instance, when such a cicatrix on the cheek draws the lower eyelid down, causing ectropion.

You will occasionally see it asserted that the cicatrization of a granulating surface may sometimes begin from several patches of epidermis forming in its midst. This is only true of cases where portions of cutis with rete Malpighii have remained in the midst of the wound, as may readily happen in gangrenous wounds, as the caustic agent may penetrate unequally deep. Under such circumstances, epidermis again forms from some remaining portion of the papillary layer, that has the slightest possible covering of cells of the rete Malpighii; at these points we have the same circumstances as when we have raised a vesicle on the skin by cantharides, inducing by the rapid exudation an elevation of the epidermis from the mucous layer of the skin; this is followed by no granulations, if you do not continue to irritate the surface, but horny epidermis again forms at once over the mucous layer. But, if there be no such remnant of rete Malpighii, we never have these islands in the cicatrix, the formation of epidermis only takes place gradually, from the periphery of the wound toward the centre. I believe this so firmly, that I think surgeons, who say they have seen otherwise, must be mistaken in some way.

The transplantation of epi [illegible] er *Reverdin's* method also appears to me to favor the view [illegible] m is only developed from epithelium.

After having [illegible] s of the wound, the developmen [illegible] cicatrix, we must now turn again to the more minute changes by which these external symptoms are induced.

It will be simplest for us, again, to represent a relatively simple capillary net-work in the connective tissue: suppose a crescentic piece to be cut out of it from above; first, there will be bleeding from the vessels, which will be arrested by the formation of clots as far as the next branches. Then, there must be dilatation of the vessels about the wound, which is due partly to fluxion, partly to increased pressure; an increased transudation of blood serum, or an exudation, is also a necessary result of the capillary dilatation, from causes above given; the transuded serum contains some fibrogenous substance, which, by the influence of the newly-formed cells in the most superficial layers, coagulates to fibrine, while the serum, mixed with blood plasma, flows off. The vascular net-work would assume the following shape:

FIG. 4.

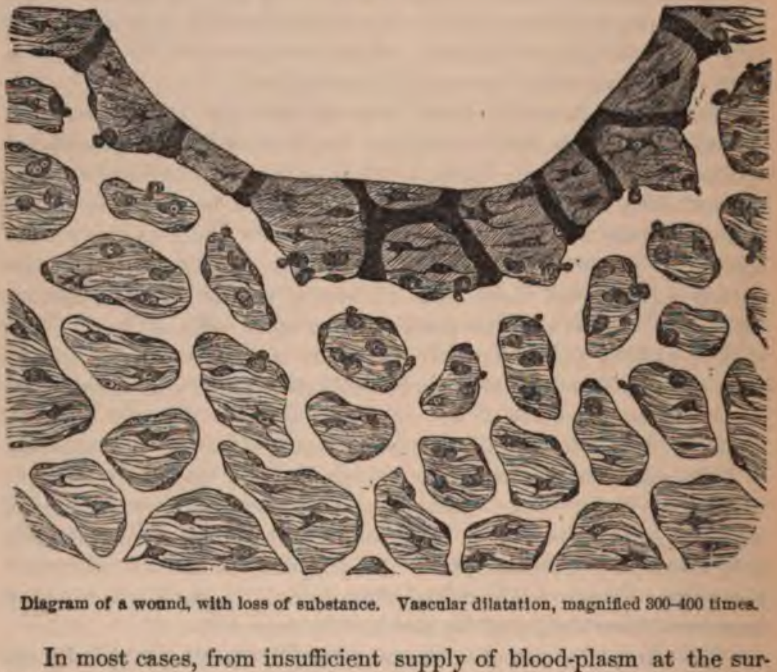


Diagram of a wound, with loss of substance. Vascular dilatation, magnified 300–400 times.

In most cases, from insufficient supply of blood-plasm at the surface of the wound, more or less particles of tissue will die; as the stoppage of vessels must, of course, deeply affect the nutrition of tissues not very vascular, and, where the tissues are very stiff, dilatation of the vessels will be interfered with. Let us suppose that the upper layer, shaded in the diagram, is dead from the changes in the circulation. What will now take place in the tissue itself? Essentially, the same changes as in the united edges of a wound; wandering of white-blood cells through the walls of the vessels, their collection in the tissue with the secondary action they induce; plastic infiltration, and inflammatory new formation. But, since there is no opposing wounded surface, with which the new tissue can coalesce, then to be quickly transformed to connective tissue, the cells, escaping from the vessels, remain at first on the surface of the wound; the exuded fibrinous material on the surface of the wound becomes soft and gelatinous; at the same time, the infiltrated tissue of the surface of the wound assumes the same peculiarities; the soft connective tissue, into which the young vessels shortly grow, even if only present in small quantities, holds together the cells of the inflammatory new formation, which constantly increase in number. The *granulation tissue* is thus formed; this is, therefore, a highly-vascular inflammatory new formation. At first, it grows constantly, the direction of its growth is from the bottom of the wound toward the surface; the tissue is, however, of different consistence in the various layers, its superficial surface especially is soft, and *most* superficially of fluid consistence, for here the intercellular substance becomes not only gelatinous, but fluid; this uppermost thin fluid layer, which is constantly flowing and being constantly renewed from the granulation tissue by cell-exudation, is *pus* (Fig. 6).

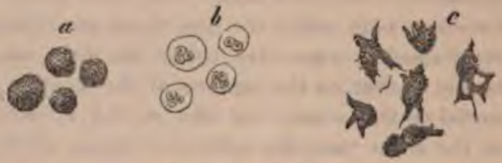
Hence, pus is fluid, as it were melted, dissolved inflammatory new formation. Where pus is present in quantity it must have come from some sort of granulation tissue or from some other highly-vascular and usually highly-cellular source; this source need not always be a surface, as in the present case, but may lie deep in the tissue and form a cavity; the centre of an inflammatory new formation anywhere in the tissue may break down into pus; then we have an *abscess.*

We shall frequently have occasion to speak of this relation of pus and granulations to each other; hold fast to the idea of granulations being tissue (not granules), and of pus being fluid inflammatory new formation, and you will hereafter readily understand many processes, especially chronic inflammations, whose variable appearance you would otherwise find incomprehensible.

Let us now say a few words about pus itself. If left standing in a vessel, it separates into an upper, thin, clear layer, and a lower yellow one; the former is fluid intercellular substance, the latter contains chiefly pus-corpuscles. On simple microscopic examination these are round, finely punctated globules, of the size of white-blood corpuscles;

they contain three or four dark nuclei, which become quite distinct on addition of acetic acid, because it dissolves the pale granules of the protoplasm, or at least swells them so that they become transparent. The nuclei are not soluble in acetic acid; the entire globule is readily dissolved in alkalies.

FIG. 5.



Pus-cells from fresh pus, magnified 400 times. *a*, dead without addition; *b*, the same cells after addition of acetic acid; *c*, various forms that living pus-cells assume in their amœboid movements.

At *a* and *b* we see the pus-cells as they usually appear when we cover a drop of pus with a thin glass, and without any addition examine it under the microscope. The above-mentioned observations of *Von Recklinghausen* have shown that only the dead cells have this round shape; if we observe the pus-cells in the moist chamber on a warmed object-table (according to *M. Schultze*), we see the amœboid movement of these cells most beautifully. These movements, which only go on slowly and sluggishly at blood-heat, become more rapid at a higher temperature, and less so at a lower. The number of pus-cells in pus is so great, that in a drop of pure pus, under the microscope, the fluid intercellular substance is not at all perceived. Chemical examination of pus is difficult, first, because the corpuscles cannot be completely separated from the fluid; also, because the large quantities of pus obtainable for chemical examination had already been a long time in the body, and may have changed morphologically and chemically; and lastly, because chiefly protein substances are contained in pus, whose perfect separation hitherto has not always been possible. If we let pus from a wound stand in a glass, the clear, bright-yellow serum usually occupies more space than the thick, straw-yellow sediment, which contains the pus-corpuscles. Pus contains ten to sixteen parts of firm constituents, chiefly chloride of sodium; the ashy constituents are about the same as those of blood-serum. Recent examinations of pus have shown that myosin, paraglobulin, protagon, fatty acids, leucin, and tyrosin, are constant constituents. Pus collected in the body does not readily undergo acid fermentation; pure fresh alkaline pus soon becomes sour, however, if it is left standing for a time even in a covered glass.

Let us now return to the granulation layer, where we have still an important point to consider, namely, the numerous vessels, which give its red appearance. The extensive vascular loops that must form on the surface of the wound, and which in the diagram (Fig. 6) are too small and too few, commence, with the growth of the surrounding granulation tissue, to elongate and become more tortuous; toward the fourth or fifth day new vessels develop as fine lateral capillary communication, as in healing by first intention, and the tissue is soon traversed by an excessive number of vessels, which have so much effect on the appearance of the entire granulation surface that it is hardly recognizable on the cadaver, where the fulness of the vessels is wanting, or is at least less marked than during life, and the tissue consequently appears pale, relaxed, and much less thick. The question arises, Whence come these remarkable, small, gradually-confluent red nodules, which are visible to the naked eye? Why does not the surface look even? Indeed, this is frequently the case; the granules are by no means always distinctly defined; but it is not easy to explain the cause of their form. It is usually assumed that the granules are to be regarded as imitations of the cutaneous papillæ; but, independent of the fact that it is incomprehensible why such structures should be imitated in muscle and bone, and that the granules are usually ten times as large as the cutaneous papillæ, this is no real explanation. The appearance of the granules, doubtless, depends on the arrangement of the vascular loops into tufts, on certain boundaries between the different groups of vessels. Hence we might suppose that the vascular loops acquire this form without known cause. Still, it seems to me natural to compare them to the circumscribed capillary districts, already formed in the normal tissues, of which we have numerous examples, especially in the skin and in fat. You know that every sweat and sebaceous gland, every hair-follicle and fat-lobule, has its nearly-closed capillary net-work, and, by the enlargement of such capillary net-works, the peculiar closed vascular forms of the granules might arise. In fact, in the cutaneous and fatty tissue you will find the individual fleshy growths, particularly sharply and clearly defined, while this is more rarely the case in muscle, where these bounded capillary districts do not occur. It can only be decided by artificial injections of fresh granulations, whether this explanation is correct; till then, it remains simply an attempt to refer this pathological new formation to normal anatomical conditions.

The following sketch, in which, on account of the great enlargement, and the small injured district, nothing can be seen of the granular layer, may serve you as a diagram of the development of the granulation tissue with its vessels, and of its relation to pus and to the subjacent matrix, as it has developed from Fig. 4.

If the growth of the granulations was not arrested at some point, a constantly-growing granulation tumor would be formed. Fortunately, this is never or very rarely the case. You already know, from the representation of the external conditions, that when the granulations have reached the level of the cutis, or even sooner, they cease to grow and are coated with epidermis, and retrograde to a cicatrix. The following changes occur in the tissue: At first, in the granulation tissue, as in the edges of the wound in healing by the first intention, there are numerous cells which are destroyed. Not only the millions of pus-cells on the surface, but also cells in the depths of the granulation tissue, disappear by disintegration and reabsorption; it is very probable that cells from the granulation tissue may pass back uninjured into the vessels, as we shall see when treating of the organization of thromboses of the vessels. As the cells retrograde, fine fat-granules gradually form in them, not only in the round but also in the spindle-shaped ones; such cells, which are compcsed of very fine

FIG. 6.

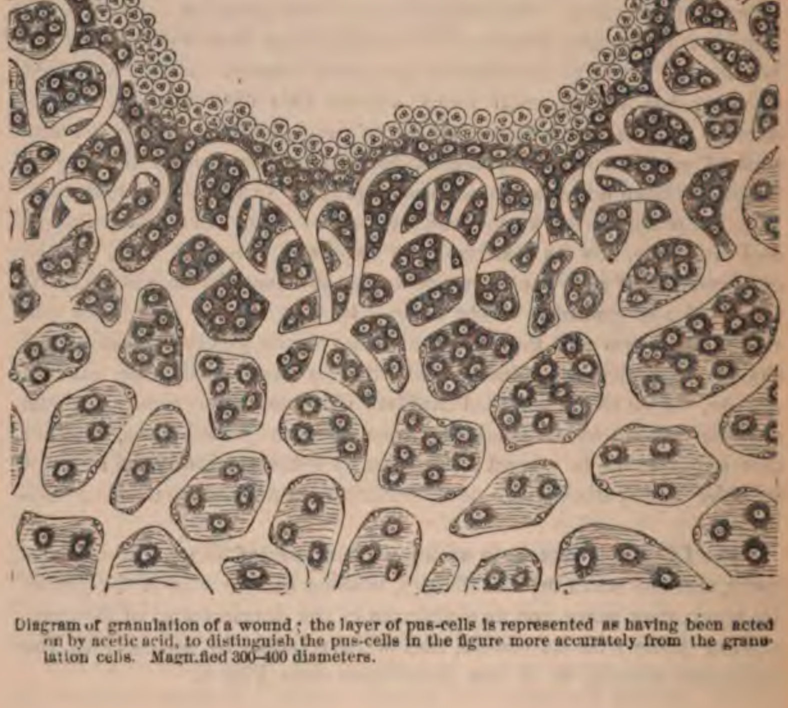
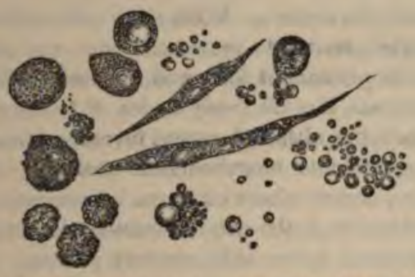


Diagram of granulation of a wound; the layer of pus-cells is represented as having been acted on by acetic acid, to distinguish the pus-cells in the figure more accurately from the granulation cells. Magnified 300–400 diameters.

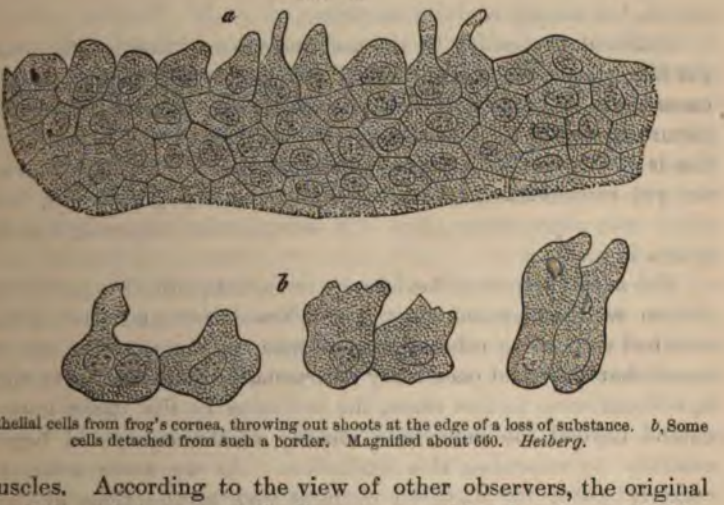
fat-globules, are generally called *granular cells* (Körnchenzellen); they often occur in the granulations, as above described. When the granulation tissue is thus diminished by atrophy and escape of the cells, and at the same time the new formation of cells ceases, something important must happen, that is, the gradual consolidation of the gelatinous intercellular tissue to striated connective tissue, which is brought about by the steadily increasing loss of water, that is carried off by the vessels and evaporated from the surface; then the remaining cells at once assume the shape of the ordinary connective-tissue corpuscles. According to the view of other observers, the original intercellular substance entirely disappears, and its place is supplied by the protoplasm of granulation cells, which transforms into fibrous tissue. With these changes which take place from the periphery toward the centre, the secretion of pus on the surface ceases; at the very

Fig. 7.



Fatty degeneration of cells from granulations. Granulation-cells. Magnified about 500 diameters.

Fig. 7 a.



*a*, Epithelial cells from frog's cornea, throwing out shoots at the edge of a loss of substance. *b*, Some cells detached from such a border. Magnified about 660. *Heiberg.*

circumference of the wound on the condensing granulation tissue epidermis forms and quickly separates into hard epidermic and mucous layers; according to *J. Arnold*, this formation takes place by the division of a protoplasm, at first entirely amorphous, in the immediate vicinity of the existing border of epidermis. Lastly, the superfluous capillaries must be obliterated; few of them remain to keep up the circulation through the cicatrix. With their obliteration the tissue becomes drier, tougher, contracts more and more, and often the cicatrix does not acquire its permanent form and consistence for years.

The whole process, like all these modes of healing, contains much that is very remarkable, although recent investigations have explained many of the more minute morphological changes. The possibility, nay, the necessity, under otherwise normal circumstances, of arriving at a typical termination, is the chief characteristic of those new formations that are induced by an inflammatory process. If this natural course of healing does not take place, it is because either constitutional or local conditions indirectly or directly interfere, or because the organ attacked is so important to life, the disturbance to the entire body so severe, that there is death of the organ, or of the individual, or that the functional disturbance of the former causes the death of the latter. Every new formation, due to inflammation, always has the tendency to reach a certain point, to retrograde, and pass into a stationary state, while other new formations have no such natural termination, but usually continue to grow.

Different as healing by the first and second intentions appears, at the first glance, the morphological changes in the tissue are in both cases the same; you only need to divide Fig. 3 at *a*, to have the same picture as in Fig, 6. Observation teaches in the simplest manner that this is actually so; if a wound almost healed by first intention, but not yet consolidated, be torn open, we have a granulating wound which soon suppurates. You will hereafter be frequently convinced of this in practice.

The above process of healing by immediate adhesion and by granulation we have termed *traumatic inflammation*, and have found it identical with some other forms of inflammation; it has also been stated that a marked peculiarity of traumatic inflammation is, that in it, without some further cause, the irritation in the tissue does not extend beyond the immediate vicinity of the injury. I beg you carefully to remember this limitation. As we know nothing accurately about the chemical changes and nerve-actions in the inflamed tissue, while we do know the morphological processes very accurately, we naturally attend most to the latter if we wish to define and generalize the term "inflammation." I will briefly take up the previous views on this subject (p. 53). "Inflammation" is a modification of the normal physiological processes in the different tissues of the body, a "disturbance of nutrition" (*Virchow*) whose histopoëtic results you now know and of whose destructive, deleterious actions you will hereafter hear. Any part of the body was said to be "inflamed" when it was hot and red; as it is then generally swollen and painful also, this name is applied to processes where the above symptoms occur. The word inflammation originated when there were no true pathologico-anatomical ideas; even the oldest observers understood that something unusual was going on in the tissues, that they were much heated (*inflammatio*), and from the first this process has been regarded as an intense increase of the vital processes. As they could not understand the process itself any better than we do, they considered the symptoms and the results of the process, just as we do; so that doubts often arose if it were proper to speak of inflammation when one or other symptom was absent or not well marked, just as it is to-day. We now know that inflammation is not an existence outside of the body, which makes its way into some part and there grows, and must be expelled like Beelzebub, and we know why "tumor, rubor, calor, dolor," are caused by inflammation, but although any one usually recognizes an acute inflammation as such and designates it correctly, it still remains difficult as well clinically as anatomico-pathologically to give an exact definition of "inflammation." There is no difficulty in distinguishing an oak from an ass; but, if you attempt to generalize and give a sharp definition between plants and animals, you will have the greater difficulty the more you know of the details of botany and zoology. The word "inflammation" is in use, and so accurately designates those processes to which it was first applied, that it would be useless to try to root it out. By it we understand the above-described combination of processes in the tissues, which in the present case arise from a purely mechanical irritation (wound) acting only once. How much hyperæmia, exudation, and new formation of tissue is required before we can term the process inflammation cannot be stated absolutely. It seems to be agreed by surgeons and anatomists to designate as "inflammatory" the *purely regenerative processes*, that is, the neoplastic tissues, which directly or indirectly replace the loss of substance. If we consider the process in the modern histological sense, it cannot be accurately defined from the inflammatory, slight as it may be occasionally. From a purely clinical point of view, the distinction is easier, as we often meet cases without any of the four cardinal symptoms on the edges of the wound; and still the difference between a slight redness, swelling, and sensitiveness of the borders of the wound to an intense, progressing inflammation over the entire affected portion of the body is only one of degree. Custom has here made a distinction; when a wound heals without any symptoms of so-called reaction we do not call it inflammation of the wound, but only apply this term when the symptoms of inflammation are very prominent at the part injured.

I deemed it necessary to speak to you of these general considerations on inflammation, so that you might early learn some of the difficulties of the subject. In these lectures it will always be my object to explain to you, as clearly as is now possible, the anatomico-physiological disturbances, and at the same time to show you histologically the origin of the clinical descriptions and expressions now in use. This is the only way we can truly ground our knowledge; without understanding this you would always be feeling around the outside of symptoms, and by clinging to certain ones fall into incurable dogmatism, which in a country doctor the world calls "narrow-mindedness," in the eminent city physician "infallibility." As the great majority of men are stupid in physical matters, you are sure even with the latter peculiarities of attaining great practical success, but you must then renounce all idea of appreciating or advancing the progress and development of society.

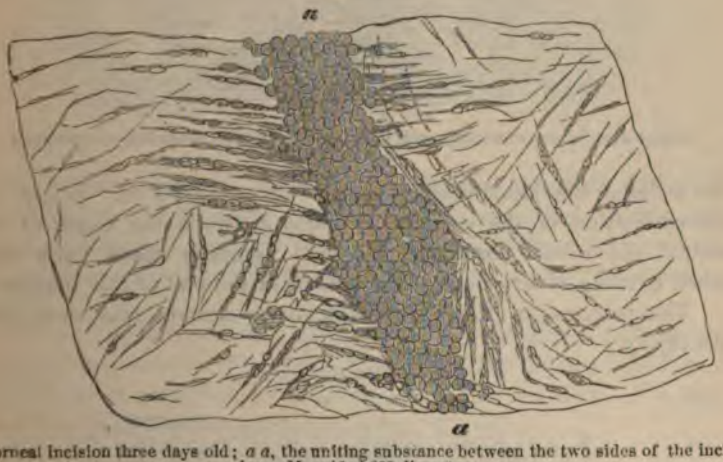
It is not the object of these lectures to show you on preparations, step by step, the morphological microscopical changes in wounded tissue—you will see these, in the practical lessons on pathological histology—but I will show you a few points, so that you may not think that the processes of which I have spoken can only be demonstrated on diagrams.

The cell-infiltration of tissue, after irritation by an incision, is best seen in the cornea. Four days ago I made an incision, with a lance-shaped knife, in the cornea of a rabbit; yesterday the incision was visible as a fine line with milky cloudiness. I killed the animal carefully, cut out the cornea, and let it swell in pyroligneous acid, till this morning; then made a section through the wound, and cleared it up with glycerine.

Now, at *a a* (Fig. 8), you may see the connecting substances between the edges of the wound, in which there has been a considerable collection of cells, between the lamellæ of the cornea, where the corneal corpuscles lie. These cells are not so evident in the method employed as in that where carmine is used, still the intermediate substance between the edges of the wound is very distinct. As you see, it consists almost entirely of cells; the cells alone would not, however, render the union sufficiently firm, if they were not glued together by a fibrinous cement. The young cells probably come out of the edges of the wound from the fissures between the corneal lamellæ, and probably do not originate in the connective substance between the edges of the wound; on the contrary, the latter is finally formed from them. Let me remark incidentally, these fine corneal cicatrices subsequently clear up, so as to leave scarcely a trace. All the cells that you here see in the preparation come from the vascular loops of the conjunctiva; the normal stellate corneal cells are not visible here. I have chosen this specimen because the intermediate substance is broad and very rich in cells. In very small incisions, made in the cornea with a very sharp knife, the intermediate substance is so slight, that it is seen with difficulty; then, too, the changes on the edges of the wound are slighter than here, and so slight a scar is not visible to the naked eye.

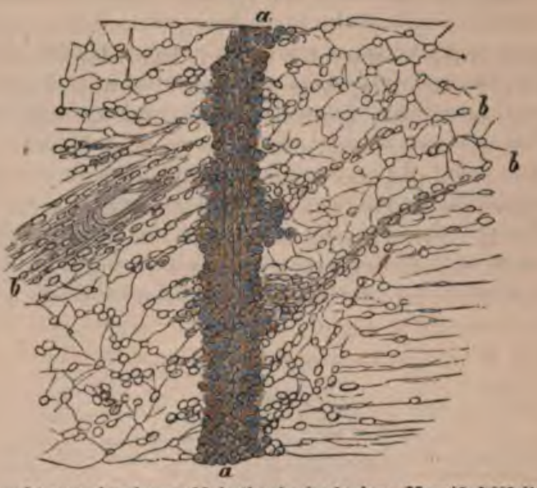
Here (Fig. 9) you have a transverse section through a twenty-four-hour old, freshly-united wound in the cheek of a dog. The incision is well marked at *a a;* the edges of the wound are separated by a dark, intermediate substance, which consists partly of white cells, partly of red corpuscles—the latter belong to the blood, escaped between the edges of the wound, after the injury; the connective-tissue fissures crossed by the wound, in which the connective-tissue cells lie, are already filled with numerous newly-formed cells, and these cells have already pushed into the extravasated blood between the edges of the wound. The preparation has been treated with acetic acid, hence you no longer see the striation of the connective tissue, but see the young cells more distinctly. Look particularly at certain strings, rich in cells, that extend from the wound toward both sides (*b b b*); these are blood-vessels in whose sheaths many cells are infiltrated; this is apparently because here many white-blood cells have passed throu

FIG. 8.



Corneal incision three days old; *a a*, the uniting substance between the two sides of the incision. Magnified 300 diameters.

FIG. 9.

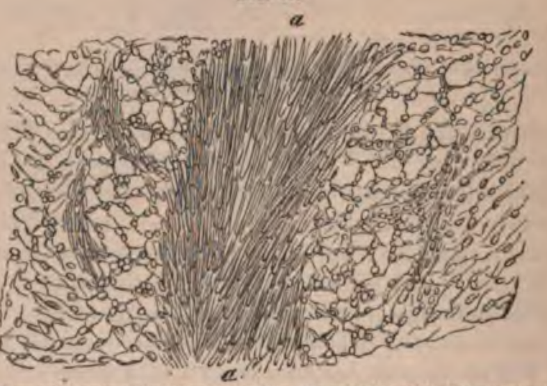


Incised wound twenty-four hours old, in the cheek of a dog. Magnified 300 diameters.

the walls of the blood-vessels, or are about to do so. About the transformation of the coagulated blood between the edges of the wound, the wound thrombus, we shall hereafter speak more fully when treating of cicatrices of the vessels at the end of this chapter.

This preparation (Fig. 10) shows a young cicatrix, nine days after the injury.

FIG. 10.

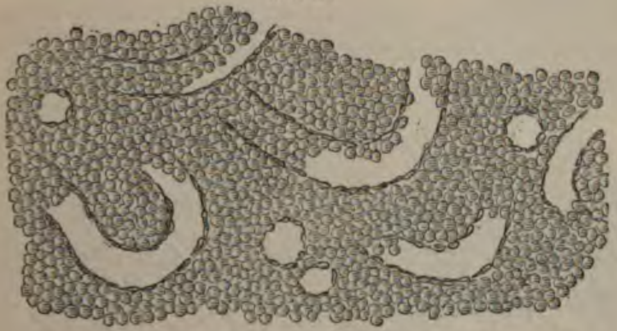


Cicatrix nine days after an incision through the lip of a rabbit, healed by first intention. Magnified 300 diameters.

The connective substance (*a a*) between the edges of the wound consists entirely of spindle-cells pressed together, which are most intimately connected with the tissue on both sides of the wound.

Fine sections cannot be made of granulation tissue, just taken from a wound; it is generally a very difficult subject for fine preparations. If you harden the granulation tissue in alcohol, color the section with carmine, then clear it up with glycerine, you have a specimen like Fig. 11.

FIG. 11.

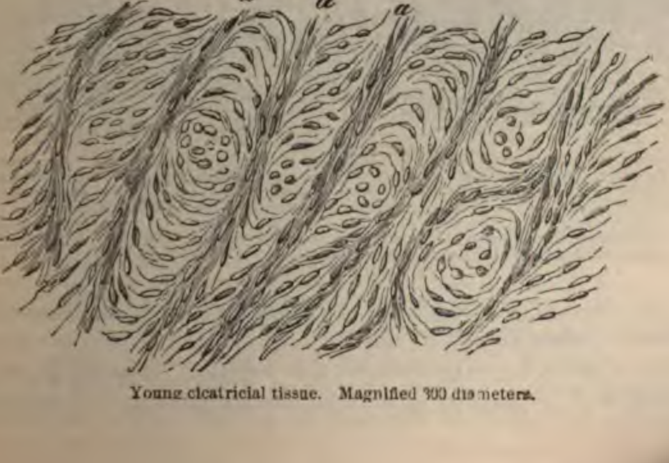


Granulation tissue. Magnified 300 diameters.

The tissue appears to consist solely of cells and vessels, with very thin walls; the whole tissue is shrunken by the alcohol, so that we here see nothing of the mucous intercellular substance which is always present, even if only in small quantities, in healthy, fresh granulations.

We see the tissue of the young cicatrix particularly well in the following preparation (Fig. 12), which was taken from a broad cicatrix, following granulation and suppuration, in the back of a dog, about four or five weeks after the injury.

FIG. 12.

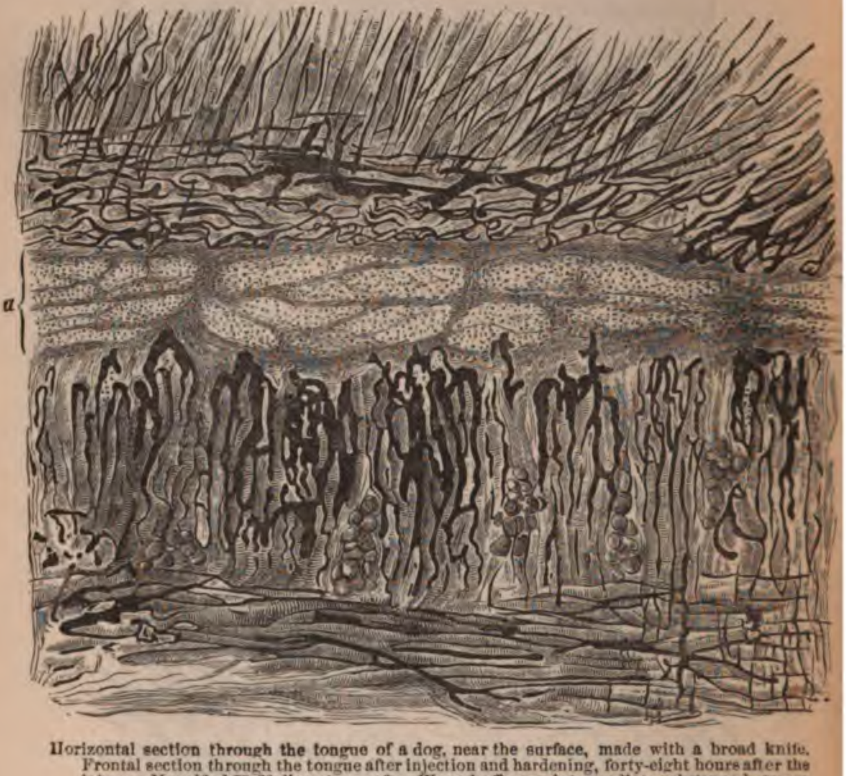


Young cicatricial tissue. Magnified 300 diameters.

The preparation has been treated with acetic acid, to show the arrangement of the connective-tissue cells, that have formed from the granulation tissue; *a a a* are partly obliterated, partly still permeable blood-vessels; the connective-tissue cells are still relatively large, succulent, and distinctly spindle-shaped, still the intercellular substance is richly developed.

To study the state of the blood-vessels in the wound, we must make injections; this is quite difficult, and quick success often depends on a lucky chance.

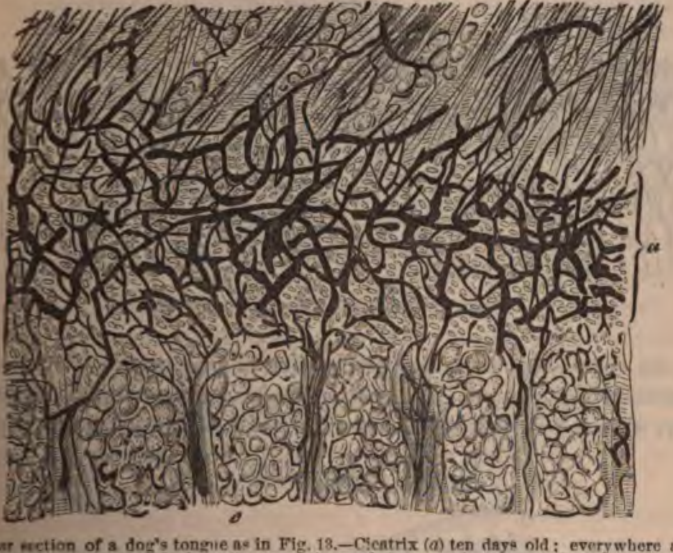
FIG. 13.



**Horizontal section through the tongue of a dog, near the surface, made with a broad knife.** Frontal section through the tongue after injection and hardening, forty-eight hours after the injury. Magnified 70-80 diameters; after *Wywodzoff*—*a a*, intermediate substance between the edges of the wound (consisting of filamentary-looking adhesive material and extravasated blood). The section has passed through two layers of muscle crossing each other. Looping of the vessels with dilatation in both borders of the wound; commencing elongation of the loops into the connective substance.

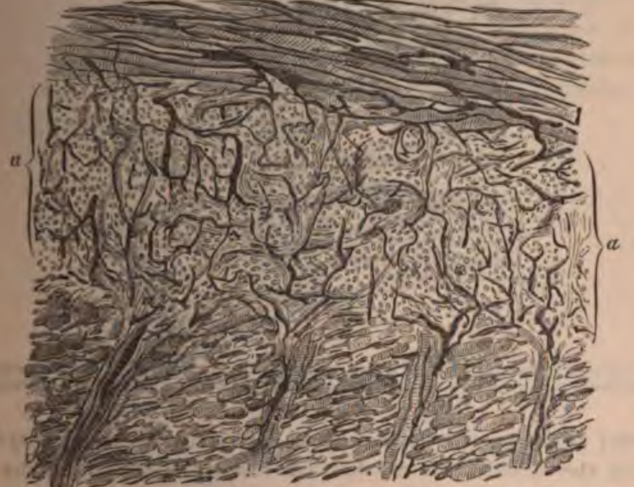
On this subject we have the recent works of *Wywodzoff* and *Thiersch*, whose results in the main agree partly with one another, partly with my investigations on this subject. *Wywodzoff*, who operated on dogs' tongues, gives a series of representations of the condition of the blood-vessels in various stages of healing of the wound, a few of which I shall demonstrate to you, without, however, going into the more minute details of the formation of vessels.

FIG. 14.



Similar section of a dog's tongue as in Fig. 13.—Cicatrix (*a*) ten days old; everywhere anastomoses of the vessels from the two edges of the wound. Magnified 70-80 diameters; after *Wywodzoff*.

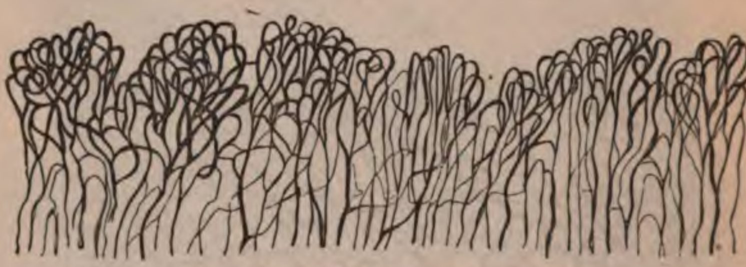
FIG. 15.



Similar section of a dog's tongue as in Fig. 13.—Cicatrix (*a*) sixteen days old. The vessels already greatly diminished and atrophied. Magnified 70-80 diameters; after *Wywodzoff*.

This (Fig. 16) is a preparation of granulations from a human being, where the vessels were tolerably filled by natural injection; the vascular loops are very close together and complicated at the surface; deep down the vessels run nearly parallel.

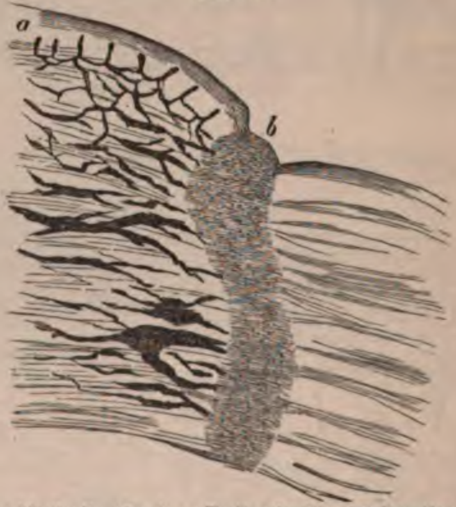
FIG. 16.



Granulation vessels. Magnified 40 diameters.

In conclusion, here is a preparation of injection of the lymphatic vessels of a dog's lip. You see that the young cicatrix, on the seventh day, when it still consists almost exclusively of cells, has no lymphatic vessels; these cease immediately at the young cicatrix; they do not form in the cicatrix till the fibrillar connective-tissue bundles form. The granulation tissue also has no lymphatic vessels; where the in-

FIG. 17.



Seven-days-old wound in the lip of a dog. Healing by the first intention. Injection of the lymphatic vessels: *a*, mucous membrane; *b*, young cicatrix. Magnified 20 diameters.

flammatory new formation, where the primary cellular tissue forms, the lymph-vessels are mostly closed, partly by fibrous coagulations, partly by new cell-formations. These observations have also been confirmed quite recently by *Lösch*, of St. Petersburg, by examinations of traumatically inflamed testicles.

[To understand the difference between healing by first intention and by granulation or second intention, you must remember that in the former the cellular cicatricial tissue directly caused the union of the edges of the wound, and subsequently changes to connective tissue. In healing of a wound with loss of substance direct union is impossible ; the cicatricial tissue must first replace the lost substance and fill up the space between the edges of the wound. Many text-books speak of healing by second intention simply as healing by suppuration ; in many cases this name does not apply ; it may take place without inflammation and consequently without suppuration, under a crust formed of dried primary secretion from the wound. But, even when inflammation occurs, it does not necessarily result in suppuration. In most cases, however, there is suppuration with moderate inflammation, resulting in granulation tissue, with such an escape of white blood-cells that many of them, especially the superficial layer, have not time to organize to firm tissue, but mingle with the secretion of the wound to form pus.

The wound may be covered with a crust of blood or primary secretion from the wound ; for healing to occur under the crust the primary secretion must be scanty and the conditions favorable for the plasmatic circulation, so that too much tissue shall not die on the surface of the wound ; moreover, all external influences causing active inflammation must be excluded so that formation of new cells may not exceed a certain point. If the cell-infiltration be too active, and plasmatic exudation from the vessels too copious, there will be suppuration and detachment of the crust.]

# LECTURE VIII.

General Reaction after Injury.—Surgical Fever.—Theories of the Fever.—Prognosis.—Treatment of Simple Wounds and of Wounded Persons.—Open Treatment of Wounds.

GENTLEMEN: You now know the external and internal minute processes in the healing of wounds, so far as it is possible to follow them with our present microscopes.

Of the *wounded person* we have not yet spoken. If you have critically examined his condition, you will have noticed changes, which may not be explained by cell-knowledge (mit Zellenweisheit), and perhaps not at all.

Possibly even the first day the patient may have been restless toward evening; he may have felt hot, thirsty, with no appetite, some headache, wakeful at night, and dull the next morning. These subjective symptoms increase till the evening after the next day. If we feel the pulse, we find it more frequent than normal, the radial artery is tenser and fuller than before; the skin is hot and dry; we find the bodily tempertaure elevated; the tongue is coated and readily becomes dry. You already know what ails the patient—he has fever. Yes, he has fever; but what is fever? whence comes it? what connection is there between the different remarkable subjective and objective symptoms? But do not ask any more questions, for I can scarcely answer those already proposed.

By the name "fever" we designate the combination of symptoms which, in a thousand different shapes, almost always accompanies inflammatory diseases, and is generally apparently due to them. We know its duration and course in various diseases; still, its nature is not fully understood, although it is better known than formerly.

The different fever symptoms appear with very variable intensity. Two of these symptoms are the most constant, viz., the increase of pulse and bodily temperature; we can measure both of them, the first by counting, the latter by the thermometer. The frequency of the heart's beat depends on many things, especially on psychical excitement of all sorts; it shows slight differences in sitting, lying, standing, walking. Hence, there are many things to which we must attend, if we would avoid error. However, we may avoid these mistakes, and for centuries the frequency of the pulse has been used as a measure of fever. Examination of the pulse also shows other things important to be known: the amount of the blood, tension of the arteries, irregularity of the heart-beat, etc.; and it should not be neglected even now that we have other modes of measurement of the fever. This other, and in some respects certainly better, mode of measuring the amount and duration of the fever is determination of the bodily temperature with carefully-prepared thermometers, whose scales are divided, according to *Celsius*, in one hundred degrees, and each degree in ten parts. The introduction of this mode of measurement into practice is due to *Von Bärensprung*, *Traube*, and *Wunderlich;* it has the advantage of graphically presenting the measurements, which are usually made at 9 A. M. and 5 P. M., as curves, and making them at once easily read.

A series of observations of fever in the normal course of wounds shows the following points: traumatic fever occasionally begins immediately after an injury, more frequently not till the second, third, or fourth day. The highest temperature attained, although rarely, is 104.5° F.–105.5°; as a rule it does not rise much above 102°–103°. Simple traumatic fever does not usually last over a week; in most cases it only continues from two to five days; in many cases it is entirely absent, as in most of the small superficial incised wounds of which we spoke above. Traumatic fever depends entirely on the state of the wound; it is generally of a remitting type; the decline may take place rapidly or slowly.

From these observations we should naturally suppose the fever would be the higher the more severe the injury. If the injury be too insignificant, there is either no fever or the increase of temperature is so slight and evanescent as to escape our modes of measurement. It has been thought that a scale of injuries might be constructed, according to which the fever would last a longer or shorter time, and be more or less intense, in proportion to the length and breadth of the wound.

This conclusion is only approximately correct, after making very considerable limitations. Some persons become feverish after very slight injuries; others do not, even after severe ones. The cause of this difference in the occurrence of traumatic fever depends partly on whether the wound heals with more or less inflammatory symptoms, partly on unknown influences. We cannot avoid the supposition that purely individual circumstances have some influence: we see that, from similar injuries, one person will be more disposed to fever than another

Before going on to examine how the state of the wound is related to the general condition, we must examine the latter a little more carefully. The most prominent and physiologically the most remarkable symptom of the fever is the elevation of the temperature of the blood, and the consequent increase of the bodily temperature. All the modern theories of fever turn on the explanation of this symptom. There is no ground for supposing that in fever any absolutely new element must be added to the requirements acting for the preservation of a constant temperature in the body, but it is probable that the fever temperature is caused by some change of the normal requirements of temperature, which vary readily with circumstances. When you remember that men and animals in the varied temperatures of summer and winter, in hot and cold climates, have about the same temperature of the blood, you will see that the conditions of production and giving off of heat are susceptible of great modification, and that within these conditions there may very possibly be abnormities of the resulting bodily temperature. It is evident *a priori* that an increase of bodily temperature may depend either on diminution of the amount of heat given off, the production remaining the same, or on increased production, the loss of heat remaining the same (other relations of these factors to each other are possible, but I shall pass over them, to avoid confusing you on this difficult question). The decision of this cardinal question does not seem possible at present; it would be possible by determining and comparing the quantity of heat produced in fever and in normal conditions, by the so-called calorimetrical experiments on men and large warm-blooded animals; but hitherto there have been great difficulties in the way of these experiments. *Liebermeister* and *Leyden* have invented methods of calorimetry, that seem to me correct; but the methods and conclusions of *Liebermeister* have been energetically combated by *Senator*. Hence, in regard to the above questions, we are still, to a great extent, thrown on probability and hypothesis. As the production of heat depends chiefly on oxidation of the constituents of the body, increase of the latter would necessarily be followed by increase of the former if the loss of heat remained the same. Now, since the amount of urea is regarded chiefly as the result of the burning up of the nitrogenous bodies, and as the amount of urea excreted in fever is usually increased, and the weight of the body rapidly decreases, as appears from the experiments of *O. Weber*, *Liebermeister*, *Schneider*, and *Leyden*, this, with the above-mentioned calorimetric experiments, is considered strong proof that in fever the consumption is greatly increased, and that consequently more warmth is really produced than in the normal state, more than can be disposed of by the body in the same time. *Traube* gives another view of the occurrence of fever-heat: he asserts that every fever begins with energetic contraction of the cutaneous vessels, especially of the smaller arteries, and that thus the giving off of heat to the air is decreased, and more heat collected in the body, without its actually producing more. Although this hypothesis is advanced by its author with wonderful ability and acuteness, and is apparently supported by the work of *Senator*, I, with most pathologists, cannot agree with it, especially as the premises, the contraction of the cutaneous vessels, can only be acknowledged in the cases begining with chill; but this chill is by no means a constant symptom in the fever. Hence, in what follows, we shall start from the point that in fever there is increased production of heat. Then arises the question, How does inflammation generally, and traumatic inflammation particularly, effect the increase of bodily temperature? This question is answered in various ways:

1. At the point of inflammation, as a result of the lively interchange of tissue, heat is produced; the blood flowing through the inflamed part is warmed more, and distributes the abnormal amount of heat here acquired, to the whole body. That the inflamed part is warmer than the non-inflamed is readily proved, especially in superficial parts, as in the skin, but this does not prove that more warmth is produced here than is usual, but is probably simply due to the circulation of more blood through the dilated capillaries; if the inflamed part be not warmer than the blood flowing to it, it is not probable that it should produce heat. The investigations on this point are numerous and contradictory. The thermometrical measurements of *O. Weber* and *Hufschmidt* have given various results; usually the temperature in the wound and in the rectum (which has about the warmth of arterial blood) were equal; occasionally the former was higher than the latter, sometimes the reverse; the difference was never great, not being more than a few tenths of a degree in any case. Recently *O. Weber* has hit on a new method of measurement, the thermoelectric: by his very difficult investigations the question seemed to be decided that the inflamed part is always warmer than the arterial blood; indeed, that the venous blood coming from the seat of inflammation is warmer than the arterial blood going to it. Quite recently these investigations were repeated in Konigsberg by *H. Jacobson*, *M. Bernhardt*, and *G. Laudien*, with the final result of showing no increase of warmth in the inflamed part. From the contradiction of the results of observation it is impossible to form a judgment on this point. Nevertheless it seems certain that in the inflamed part there is not enough heat produced to elevate the temperature of all the blood in the body several degrees.

2. The irritation induced by the inflammation on the nerves of the inflamed part might be supposed as advancing to the centres of the vasomotor (nutrient) nerves; the excitement of the centres of these

nerves would induce increase of the general change of tissue and consequent increase of the production of warmth. This hypothesis, which is supported by some facts, such as the great difference in febrile irritability, and which I formerly maintained, no longer appears to me tenable; it is opposed by the experimental researches of *Breuer* and *Chrobak*, which prove that fever occurred even when all the nerves were divided, by which there could be any conduction from the peripheral injury to the nerve-centres; the recent investigations of *Leyden* also oppose this hypothesis, since they prove that there is no constant relation between the loss of nitrogenous material, or consumption, and development of warmth.

3. Since, from the nature of the process, in the inflamed part some of the tissue is destroyed, while some new tissue is formed, it is not improbable that some of the products of this destruction enter the blood, partly through the blood-vessels, partly through the lymph-vessels; such material acts as a ferment, excites change in the blood, as a consequence of which the entire amount of blood may be warmed. We might also admit a more complicated mode of development of warmth, which, by including the nervous system, might in some respects be more serviceable theoretically; the blood changed by taking up the product of irritation might prove irritant to the centres of the vaso-motor nerves, and thus induce increased production of warmth. The decision between these different hypotheses is difficult; they are all about equally justifiable, and all have the common factor of pollution of the blood by material from the seat of inflammation or the wound, which is recognized as having an effect on the production of heat; these substances must have the effect of exciting fever (a *pyrogenous* action). This was to be proved. It has been proved by experiments of *O. Weber* and myself, which I can notice only briefly here. In most open wounds, especially in contused wounds, shreds of tissue are always decomposed; in many idiopathic inflammations, the circulation is arrested at different points in the inflamed tissue, and there is partial decomposition of these dead portions. Decomposing tissue, then, was an object to be examined in regard to its pyrogenous action. If you inject filtered infusions of this substance into the blood of animals, they have high fever, and not unfrequently die with symptoms of debility, of somnolence, with coincident bloody diarrhœa. The same effect is induced by fresh pus injected into the blood; a weaker effect follows the employment of juice and pus serum pressed out of the inflamed part, but the secretion from the wound taken during the first forty-eight hours is especially active. Hence the products of decomposition, as well as those of new formation, have a pyrogenous action in the blood. These products are of a very complicated and variable nature; some of the

chemical substances in them have been independently tested in regard to their fever-exciting qualities: we may induce fever by injecting leucin, sulphuretted hydrogen, sulphides of ammonium and carbon, and other chemical substances resulting from the decomposition of tissue, or even by injecting water; decomposing vegetable matter also has a fever-exciting effect. Hence there are no specific fever-exciting substances, but the number of pyrogenous materials is innumerable.

I may here mention that the bad-smelling substances developed by the decomposition of the tissues are probably the least dangerous. I intentionally distinguish the products of decomposition in acute inflammations, which are usually odorless at first (whose activity as poisons we first learned by experiment), from those of decomposing dead bodies, which generally smell bad at once, although their pyrogenous action is similar. If a wounded patient has fever, it is for me a proof that there is decomposition going on in his wound, and that the products have passed into the blood, whether the wound smells or not.

After the pyrogenous effect of the products of inflammation and decomposition had been absolutely confirmed, it remained to be proved that this material could be taken from the tissue into the blood, and to be shown how this took place. For this purpose it was injected into the subcutaneous cellular tissue, where it spread around in the meshes of the tissue; the effect, as to fever, was the same as when the injection was made directly into the blood; hence the pyrogenous material is absorbed from the cellular tissue. Here there is another observation to be made: after a time, at the point where decomposing fluid or fresh pus has been injected, there is severe and not unfrequently rapidly progressive inflammation. For instance, I injected half an ounce of decomposing fluid into the thigh of a horse; in twenty-four hours the whole leg was swollen, hot, and painful, and the animal very feverish. I did the same thing with the same result, with fresh (not decomposing) abscess pus, in a dog. This action of pus and putrefying matter in exciting local inflammation I call *phlogogenous*. All pyrogenous substances are not at the same time phlogogenous; some are more so than others, and, especially in the putrefying fluids, it makes a great deal of difference whether the poisonous power, which we do not know accurately, is present in greater or less quantities.

It is not certainly determined whether the pyrogenous materials enter the blood through the lymph or blood-vessels; they may vary in this respect. Some points are in favor of the reabsorption taking place chiefly through the lymphatics.

There is still something to be said about the *course* of the fever artificially induced in animals. The fever begins very soon, often even in an hour after the injection; after two hours there is always considerable elevation of temperature: for instance, in a dog whose temperature in the rectum was 103° F., two hours after an injection of pus it may be 105°, and four hours after the injection 107°. It is immaterial whether the substance be injected directly into the blood or into the cellular tissue. The fever may remain at its height from one to twelve hours, or even longer. The defervescence may be either gradually or by crisis. If we make new injections, the fever increases again; by repeated injections of putrefying material we may kill the largest animal in a few days. Whether an animal shall die from a single experiment, depends on the amount and poisonous qualities of the injected material in relation to the size of the animal. A medium-sized dog, after the injection of a scruple of filtered decomposing fluid, may be feverish for a few hours and be perfectly well after twelve hours. Hence the poison may be eliminated by the change of tissue, and the disturbances induced by its presence in the blood may again subside.

I will now terminate these observations, and only hope I may have made this important subject, to which we shall frequently return, comprehensible to you. I feel convinced that traumatic fever, like any inflammatory fever, essentially depends on a poisoned state of the blood, and may be induced by various materials passing from the seat of inflammation into the blood. In the accidental traumatic diseases we shall again take up this question.

Now a few words about the prognosis and treatment of suppurating wounds.

The *prognosis* of simple incised wounds of the soft parts depends chiefly on the physiological importance of the wounded part, both as regards its importance in the body and as regards the disturbance of function in the part itself. You will readily understand that injuries of the medulla oblongata, of the heart, and of large arterial trunks lying deep in the cavities of the body, should be absolutely fatal. Injuries of the brain heal rarely; the same is true of injuries of the spinal medulla—they almost always induce extensive paralysis and prove fatal by various secondary diseases. Injuries of large nervous trunks result in paralysis of the part of the body lying below the seat of injury. Openings into the cavities of the body are always very serious wounds; should they be accompanied by injury of the lung, intestines, liver, spleen, kidney, or bladder, the danger increases; many of these injuries are absolutely fatal. Opening of the large joints is also an injury which not only often impairs the function of the joint, but is often dangerous to life from its secondary effects. External circumstances, the constitution and temperament of the patient, have also a certain influence on the course of cure. Another source of danger is the accessory diseases which subsequently arise, and of which unfortunately there are many; of these we shall hereafter speak in a separate chapter. You must for the time being content yourselves with these indications, whose further elucidation forms a very considerable part of clinical surgery.

We may give the *treatment* of simple incised wounds very briefly.

We have already spoken of the uniting of wounds without loss of substance, and the proper time for removing the sutures, and that is about all that we can regard as directly affecting the process of healing. Still, as in all rational therapeutics, here it is most important: 1. To prevent injurious influences that may interfere with the normal course; 2. Carefully to watch the occurrence of deviations from the normal, and to combat them at the right time, if possible.

If we, first of all, limit ourselves to local treatment, we have no remedy for decidedly shortening the process of healing by first intention or by suppuration, say to half its time or less. Nevertheless, most wounds require certain care, although innumerable slight wounds heal without being seen by a surgeon. The first requirement for normal healing is absolute rest of the injured part, especially if the wound has extended through the skin into the muscles. Hence, in wounds at all deep, it is very necessary that the patient should not only keep his chamber, but that he should remain in bed for a time, as it is evident that the movement of injured parts, especially of injured muscles, must interfere with the process of healing. The second important point is cleanliness of the wound and its vicinity. Formerly it was always considered necessary to cover the wound, and to apply dressings in all cases. Of late I have grown doubtful if this be indeed necessary; indeed, I would go so far as to assert that in many cases it is well not to apply any dressings. In wounds that have been sewed up, it has often been observed that it does no harm to leave them uncovered. If we wish to cover sutured wounds, on account of pain, redness, and swelling, or because they are in a part of the body upon which the patient must lie, we may apply various kinds of dressing; we may smear the edges of the wound with pure, fine oil, best with almond-oil, and lay on a fold of linen dipped in oil, which should be changed daily, till the sutures are removed; or else we may apply a linen compress three or four layers thick, and the size of the wound, wet with water, and cover it with oil-silk, gutta-percha sheeting, or parchment-paper, and make a few loose turns a bandage over it.

For some time past I have used as the immediate covering of recent wounds merely a moistened thin sheet of gutta-percha, over this a moist compress; and to prevent the latter from drying, I cover it with some waterproof stuff, such as glazed paper, gutta-percha, or oiled silk, and then cover with plenty of dry wadding (deprived of fat and made bibulous by cooking in lye). This dressing may be removed without wetting or giving pain; it is to be retained in place by a bandage or adhesive plaster. For moistening the compresses and the sheet of gutta-percha, which lies directly on the wound, we generally employ liquids which arrest the decomposition of the secretion from the wound and prevent its smelling badly, that is, which are antiseptics and deodorants, and at the same time may destroy any infectious matters clinging to the dressings. In my clinic, for this purpose we employ saturated solution of chloride of lime, aqua plumbi, solutions of carbolic acid, carbolate of soda, and sulphate of soda (10 per cent.). I have not noticed any decided difference in their effect, and on the score of economy use solution of chloride of lime for ordinary dressings.

The frequency with which the dressings of a simple wound should be renewed depends on the amount of secretion. As a general rule, during the first four days the dressing above described should be removed at least twice daily; if during the first and second days the secretion escapes in a few hours, the dressing should be changed at once. In doing this we no longer need to use a syringe, and to carefully work off the charpie from the wound, while the patient suffers tortures; should it ever be necessary to inject fistulous wounds, of which we shall hereafter speak, we may use either a simple syringe or an *Esmarch's* douche, which consists of a cylindrical vessel 25 centimetres high and 12 in diameter, with a short tube inserted at its bottom, on to which a rubber tube with a nozzle is applied; when this vessel is held up by an attendant, it acts as a syringe. It is generally enough to wipe off the wound with a little wadding when changing the dressing, and it is not necessary to remove every trace of pus.

In many cases this dressing may be continued for weeks, being after a time applied only once daily, and then every two or three days; cicatrization goes on and the wound heals without doing any thing more.

Nevertheless, independent of certain diseases of the granulations, of which we shall speak more particularly hereafter, it frequently happens that under a continuance of the same treatment the healing is arrested; for days the process of cicatrization does not advance, and the granulating surface assumes a flabby appearance.

Under such circumstances it is advisable to change the dressing, to irritate the granulating surface by new remedies. These temporary arrests of improvement occur in almost every large wound. Under such circumstances you may order fomentations of warm camomile-tea; several compresses may be dipped in the warm tea, wrung out, and from time to time applied fresh to the wound; or you may prescribe lotions of lead-water. You may also paint the wound from time to time with a solution of nitrate of silver (two to five grains to the ounce of water). If the wound-surface be no longer large, you may finally make use of salves; these should be spread thinly over charpie or linen; the most suitable are the basilicon-ointment (compound resin cerate, consisting of oil, wax, resin, suet, and turpentine) and a salve of nitrate of silver (one grain to a drachm of any salve, with the addition of Peruvian balsam). If the cicatrization be already far advanced, we may employ zinc-salve (zinc. oxide ʒ j, ung. aq. rosæ ℥ j), or let the dry charpie adhere, and have the last portion of the wound heal under the scab.

A very peculiar and occasionally a very efficient method of hastening cicatrization of granulating wounds has been introduced by *Reverdin*. He found that a small portion of cutis taken from the surface of the body with concave scissors, and fastened with the raw surface on the granulations by means of adhesive plasters, not only becomes adherent, but the transplanted epidermis begins to grow and forms the centre of a so-called cicatricial island, whence the skinning over of the wound advances just as it does from the margins. In the clinic we have often resorted to this artificial skinning over of wounds with epidermis, and rarely ineffectually. The effect is perceived when we remove the plaster on the third day and find a red aureola around the transplanted piece; this gradually grows, and on the sixth or eighth day is followed by a bluish-white border, just as in cicatrization at the edges of the wound. I do not underestimate the practical value of this proceeding, but it is even more interesting to me from the addition it forms to our knowledge of natural history. Here we have the most striking proof not only of the independence of cell-life in the tissues of man, but still more of the readily-excited formative power of the epithelium, which is here aroused by a change of the nutrient material, while the portion of the papillary layer of the cutis transplanted at the same time does not grow.

*Thiersch*, *Minnich*, and *Menzel* have made observations showing that, eight hours or perhaps longer after death, epidermis may be successfully transplanted. The finer details of the histological changes in these transplantations have been carefully studied by *Reverdin*, and still more so by *Amabile*. *Czerny* has shown that mucous membrane from the mouth (with flat epithelium) and from the nose (with cylindrical ciliated epithelium) may be successfully grafted on wounds. [Is this, perhaps, one cause for animals licking their wounds?] The epithelium of these membranes preserves its character but a short time, then it is transformed into epidermis.

[March 6, 1871, Dr. *B. Howard* presented, at the meeting of the New York County Medical Society, a case in which, after skin-grafting, cicatrization had progressed for a time, then seemed to be arrested; whereupon he grafted small portions of the biceps *muscle* and thus induced a continuance of the cicatrization. The question was raised whether the renewed activity was not due to the previous *skin*-grafting. Dr. *Stein* stated that he had aroused these old ulcers by sprinkling epidermis scales over their surface.]

[It is not yet determined which of the constituents of the primary wound secretions have most pyrogenous action. We know that an injection of *leucin* may induce fever without exciting local symptoms, and it is known that leucin is contained in the lymph. It would seem that reabsorption of extravasated blood and lymph, even from subcutaneous wounds where there has been no decomposition, may cause fever; as we sometimes see in simple fractures, where a few hours after the injury there is marked elevation of temperature without any sign of inflammation. On the other hand, a compound fracture may run its course without fever, if the secretions from the wound escape readily. Hence we see, it is not the extent or severity of the wound that induces fever a few hours after the injury. Clinical observation seems to teach that reabsorption of undecomposed primary secretion may often induce considerable rise of temperature, but generally has little such effect. If we have a wound with loss of substance covered by a crust of dried secretion, and inflammation appears around it, secretion collects under the crust, raises it up, and exposes the granulating surface; if the secretion be reabsorbed the temperature will rise in proportion to the amount of pyrogenous substance taken into the circulation. Observation shows that decomposition depends on the presence of microscopic organisms, which act as ferments, and by multiplying excite decomposition. For their increase proper nourishment, moisture, and heat are requisite, and, above all, the presence of a germ brought in contact with the part by the air or otherwise. The air is the usual bearer of these germs; they may be filtered from it by cotton, or destroyed by heating to the boiling-point of water.

In cleansing a wound you wish to get rid of everything that may disturb its normal course; of course you remove all foreign bodies and coagula of blood, both because they may prevent union by first intention, and may, from contact with atmospheric germs, be infected; at the same time you wash off the primary wound secretion, as its absorption may induce fever and inflammation. Then you try to prevent the entrance of anything that may excite decomposition, by disinfecting with some fluid, such as a two-per-cent. solution of carbolic acid, which contains no ferment; then keep the parts at rest.

As the undecomposed secretion from the wound has only a temporary effect, the chief aim of modern surgery is to prevent decomposition in the wound, and as these decomposing masses are called *septic* matters the treatment used to prevent them is called *antiseptic*. Animal substances are more readily preserved from decomposition the more freely they are exposed to air; hence wounds were left wide open, so the dead tissue might dry up and not decompose; this is the open treatment of wounds. Healing by this method would be slower than under an antiseptic dressing.

*Pasteur* has found forms of vegetation that exist without oxygen and are even killed by it; to them he ascribes great activity, and claims that they are the sole cause of septicæmia. This question has not yet been fully investigated by others.

Experiments seem to prove that the *spray*, with two to five per cent. of carbolic acid, in *Lister's* dressing, is unnecessary and often injurious. It does not prevent the entrance of germs; in fact, may even carry them into the wound. Living micrococci have been found in pus from acute abscesses, and in an acute inflammation of the medulla of bones, hereafter to be described, decomposition within the bone occurs early. Infection from within could hardly be combatted by Listerism. The symptoms of poisoning by carbolic acid are those of collapse, fall of temperature, vomiting, bloody diarrhœa, green urine, etc.; the latter symptom occasionally occurs without others; the antidote is sulphate of soda given internally: it probably combines with the carbolic acid.

*Déclat*, a French physician, who published a large book on phenic acid, in Paris, 1865, claims that most of the evil effects of carbolic acid are due to impurities, the presence of cresylic acid, etc. He gives phenic acid alone, or combined with iodine, sulphur, or ammonia, in a great variety of diseases, and [illegible] m, and hypodermically.

*Volkmann* applies the antiseptic dressin [illegible]
is carefully washed with a two-per-cent. s [illegible]
drainage-tubes applied through it, sutu [illegible]
strip of gutta-percha tissue applied over [illegible]
carbolized gauze as large as a handkerc [illegible]

around the wound so as to make regular pressure, and kept in place by snugly applied bandages of carbolized gauze ; this is covered by a large piece of gutta-percha tissue and tightly bandaged. The bandage is to be removed if the secretion comes through, or special symptoms such as pain, fever, etc., develop. If there is fever, inquire about the bowels ; constipation may induce a rise.

Dr. *Starcke*, in the *Charité Annalen* of Berlin, 1882, in an article on permanent antiseptic irrigation, recommends a four-per-cent. solution of boracic acid with 0.1 per cent. thymol. He has abandoned *Burow's* solution, saying "it forms sediments and concrements in the sinuosities of the wound, and thus hinders escape of the pus." *Starcke* thinks the wash should be rather warm ; he commenced with low temperatures, but, as detachment of necrosed parts began, used the wash warmer, and found it better borne.]

*Samuel*, who in his last work on fever in general agrees in the etiology given in this chapter, and refers the increased temperature to increased irritation of those nerve-centres which cause production of warmth, denies the existence of a pyrogenous poison, and brings up some strong points against it ; he thinks that the blood-change which is the final essence of fever is always the same, in spite of the variety of forms under which it appears. To prevent the introduction and removal of blood and water, etc., from being regarded as pyrogenous actions in the broad sense, he supposes a similar decomposing tendency of the blood to exist between the products of inflammation, the above-mentioned and other influences, and the nervous centre which is to be excited ; and this is termed the true final pyrogenous factor, the essential element of fever.

Regarding constitutional treatment, we can accomplish scarcely anything with internal remedies in preventing or cutting short the subsequent fever. Still, certain dietetic rules are necessary. After the injury the patient should not overload his stomach, but, as long as he has fever, must live on low diet. This he usually does spontaneously, as fever patients rarely have any appetite ; but, even after subsidence of the fever, the patient should not live too high, but only eat as much as he can digest while lying in bed or confined to his chamber, where he has no exercise. If the fever be high, and the patient desires some change of drink from cold water, you may order acid drinks, as lemonade or some medicinal substance dissolved in water, oatmeal gruel, barley-water, etc. We may give the taste of the patient full play ; but it is well for you to attend to such things yourself. The physician should know as much about the cellar and kitchen as about the apothecary-shop, and it is even well for him to have the reputation of being a gourmand.

## LECTURE IX.

Combination of Healing by First and Second Intention.—Union of Granulation Surfaces.—Healing under a Scab.—Granulation Diseases.—The Cicatrix in various Tissues; in Muscle; in Nerve; its knobby Proliferation; in Vessels.—Organization of the Thrombus.—Arterial collateral Circulation.

To-day I have first simply to add a few words about certain deviations from the ordinary course of healing, which occur so frequently that they must very often be counted as normal; at all events, as very frequent.

It is not at all unfrequent for the two forms of healing above described, by first and second intention, to combine in the same wound. For instance, you unite a wound completely, and may sometimes observe that at some places there is healing by the first intention, while at others, after removal of the sutures, the wound gapes, and subsequently heals by suppuration.

But it is much more common for a large and deep wound to heal superficially, and to suppurate for some time from the deeper part. If the entire surface of the wound be healthy, the cause of the incomplete healing is either that it was imperfectly coapted at the first dressing, or that blood and exudation escaped between the edges, which not only do not coagulate firmly enough to keep up the adhesion, but can even decompose and set up an inflammation which may spread rapidly and cause severe general disturbances. These important results of such wounds compel us specially to study their mechanical conditions and chemical changes; from the first they are more or less complete *fistulous wounds*.

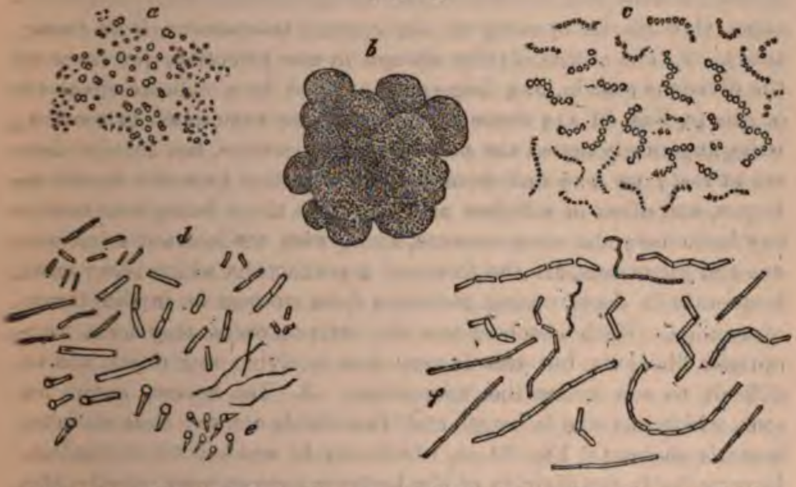
It may be readily seen that where the skin has been divided, as for the removal of a deeply-situated tumor or a portion of diseased bone, a cavity is left if the skin is sewed up, which will remain filled with air and blood, unless the bleeding has been completely arrested, the wound well cleansed, and its edges brought well in contact. In cases where different tissues are wounded, and contract unequally, as in a wound going down to the bone, the surfaces would be very uneven and not be accurately apposed if the edges of the skin were simply united. Experience teaches that in such cases large wounded surfaces, even if loosely approximated, may be readily separated by secondary hæmorrhages or fluid exudations, which often decompose while the skin above them is completely united. Then the parts around the wound swell and become painful, and high fever comes on. I will not here describe those dangerous states, septic phlegmon and blood-poisoning, which may arise, but merely say that we may often prevent the development of these processes by early evacuation of the decomposing matter. It is not the mere presence of blood between the tissues that causes these affections, for that often occurs in severe contusions without wounds and induces no bad results; it is the decomposition of the blood, and the peculiarly phlogogenous and pyrogenous properties of the first exudation, which cause the danger. Hence, in treating these wounds, we must take care, first, to prevent collection of blood and secretion in the wound, and secondly, in case this has not succeeded, to prevent decomposition of these fluids, so that they may rest quietly until absorbed, as they would if the skin had not been injured.

Of course, if there be no blood or secretion in the wound, they cannot decompose; hence it is most important to prevent their collection. This would be most simply prevented by not closing up deep wounds, but filling them with charpie, wadding, or similar bibulous material, after carefully arresting the hæmorrhage; this dressing must be renewed as often as it becomes saturated. This method was used for years, and was considered satisfactory, as no other way was known; still, as we now know better methods, we think the reaction was considerable, although less than accompanied the irritative treatment of the middle ages; inflammations spreading from the wound were frequent, and were referred to individual peculiarities, then to general influences of the atmosphere or to hospital air. It is only within the last twenty years that the propriety of the above treatment has been questioned, and new ways, based on different hypotheses, have been sought. This led to two opposite methods: one entirely without dressings (open treatment of wounds), the other accurate closure and air-tight dressing (method by occlusion). In the open treatment of wounds, which can only be used with facility in wounds of the extremities, the part is so placed that the secretion may flow readily into a vessel placed beneath. The first two days this secretion is of a dark blood-color and thin; from the third to the fifth day it becomes light brownish, then yellow, and soon in the vessel the pus-serum separates from the lumpy flakes of pus-cells; at the ordinary temperature of the room this secretion does not begin to smell badly in twenty-four hours, unless considerable quantities of decomposing dead shreds of tissue lie in the wounds and pass off with the secretion. This freedom from smell must strike any one who has smelt dressings that have been removed from a wound after being applied twenty-four hours. The bodily temperature to which this secretion is subjected while in the dressing is doubtless the cause of its more rapid decomposition. Should one *a priori* suppose that with such a dressing collection of the secretion with its evil results would be impossible, he will soon find practically that the object of the open treatment of wounds will not be attained by absolute inattention, but that the form and position of the wound may greatly impede the escape of secretion, and also that the early, firm union of the skin may shut off certain parts of the wound as effectually as if a suture had been introduced, and thus the same severe diseases may be induced as by the old methods of treatment. In operations we may do much to make wounds of such a shape that the secretions will run off at once ; but in accidental wounds this is often difficult to do, and requires a certain experience. In regard to the above-mentioned formation of pockets, we should prevent it by daily breaking up the adhesion, or from the first lay drainage-tubes in all the angles and hollows of the wound, through which any secretion from the deeper parts may readily escape. These drainage-tubes, introduced by *Chassaignac*, are made of vulcanized rubber of various calibre, with holes along the sides. The term "drainage" is taken from agriculture ; land may be drained by laying a system of porous tubes at a certain depth through the soil; the water trickles into these tubes, and flows through them to large ditches. The results from careful trial of this method of open treatment for years far surpassed all previous ones. From the publications of *Bartscher*, *Vezin*, and *Burow*, I had my attention called to this plan over ten years ago ; and as it fully agreed with the views I had arrived at from clinical and experimental observations and investigations on the poisonous peculiarities of the first secretion from wounds, I have pursued it with particular care, and have resorted to it in almost all deep wounds of the extremities, whether incised or contused. It was only after being assured by some of the most prominent German surgeons that better results were obtained by *Lister's* careful antiseptic dressing that I would try it, so little did I think of the correctness of the theory.

There is no doubt that it would be a great advantage for the patient and a triumph for surgery if we could without danger induce healing by first intention in all large deep wounds. It is true, even in the open treatment of wounds, the surfaces may so come in contact as to heal almost entirely by first intention ; but this is rare, although partial adhesions are frequent and do not require breaking up if the patient remain free from fever and pain. Formerly, by applying bandages to press the surfaces of the wound together, or by deep sutures, attempts were made to induce immediate union ; although this succeeded in some cases, it proved so dangerous in those where the surfaces of the wound were separated by blood or exudation, which putrified and could not escape, that conscientious surgeons soon abandoned it. Subsequently, when attempts were made by laying strips of oiled rag in the angle of the wound to give exit to the secretion, it rarely succeeded. In my opinion *Lister* deserves great credit for having shown that numerous drainage-tubes, properly placed in the wound and cut off even with the surface, will completely carry off all secretion, even if an accurately-fitting compressive dressing of bibulous material be applied over the united wound; if, by directly covering the wound with gutta-percha or oiled silk, we prevent the drainage-tubes from being stuck up by dried secretion, such a dressing has the advantage of an open dressing, by allowing free escape of secretion, as well as that of a compressing dressing, by which union of large wounded surfaces is so greatly favored. To prevent the escaping secretion from decomposing in the dressing and affecting the wound, the dressing should be frequently changed at first. In this care about dressings, as well as in cleanliness about operations, it seems to me, lies the great advantage of *Lister's* method. But *Lister* started on the construction of his complicated dressing from different ideas; he thought, just as I have repeatedly asserted, that the severe inflammations about wounds and the constitutional implications are almost always due to decomposition in the wound. I think that decomposition of dying tissue and exudation from the wound (for us a decomposition of albuminous substances with formation of pyrogenous and phlogogenous matters) is a chemical process that must, under certain circumstances, always occur in these substances without the addition of new agents; while *Lister* agrees with *Pasteur's* view that decomposition only occurs under the influence of small vegetable organisms, just as he claims that fermentation is only developed by yeast fungus. In regard to this question of living or dead ferments, I must refer you to organic chemistry. In physiology you have learned about salivary, pancreatic, and gastric ferments, which, although produced by cell-activity, no longer act as living organisms, but in a purely chemical way. In the same way, I think a substance may be formed as the last action of a dying tissue, that shall have some of the peculiarities of a ferment, and at the same time have a phlogogenous action, and perhaps be very poisonous for the circulating blood. It does not seem to me to have been proved that the addition of small organisms (vibriones or bacteria of *Pasteur*) is absolutely necessary to the formation of such substances. It is true, they are generally found in such fluids; but this may be explained by the fact that these small organisms occur everywhere in air and water, and develop particularly in decomposing fluids.

As we shall often have occasion to speak of these small organisms, whose significance is at present so much discussed, I will here give you a brief sketch of those forms that are most frequently found in decomposing tissues and fluids. They may be minute spheres (micrococcus, from *μικρὸς*, small, and *ὁ κοκκός*, the germ), or minute rods (bacteria, from *το βακτήριον*, the rod), which may be isolated, in pairs, or in chains of from 4 to 20 links (streptococcus, from *ὁ στρεπτὸς*, the chain, and *ὁ κοκκός*) ; often they are held in the shape of a sphere or cylinder by a glutinous substance which they throw out (coccoglia, from *κοκκός* and *ἡ γλία* or *γλοιά*, glue).

FIG. 17 A.



*a*, Micrococcus (Monads of *Hueter*. Microspores of *Klebs*); *b*, Coccoglia or Gliacoccus (Zoöglœa, *Cohn*); *c*, Streptococcus (Torula); *d*, Bacteria; *e*, Vibriones; *f*, Streptobacteria (Leptothrix of *Hallier*). Magnified 300–500

These elements vary greatly in size, from a pale sphere, of such diameter that it can scarcely be perceived with the highest power of a microscope, to the size of a pus-cell ; they are sometimes movable, at others quiet. It is pretty generally agreed that these minute organisms are vegetable in their nature, and belong to the algæ ; but their accurate botanical position and relations to each other are still matters of dispute ; their development is not yet explained, and until very recently some believed that they were the result of *generatio æquivoca* or *abiogenesis*, that is, existed without influence from any living organism. From my investigations, I think that all the above forms belong to one plant, which, being composed of coccus and bacteria, and being found chiefly in decomposing fluids, I have called *Coccobacteria septica*. This plant seems to me to develop as follows : first, its germs are found in dry air, and may be recognized under the microscope as fine dust; placed in water, they swell and throw out more or less small pale spheres, micrococcus (*a*, Fig. 21 A). According to external circumstances, these assume the following forms: 1. While increasing by segmentation, they throw out a slimy cement (*glia*), by which they hang together in balls, like frog-spawn (coccoglia, or gliacoccus, *b*, Fig. 21 A); on the surface of fluids this form often appears as coherent bright-brownish membranes, and it also grows into the interstices of tissues, and is found as whitish-gray flakes in fluids; this form is always without motion. Under certain circumstances the glia around these spheres and cylinders thickens to a membrane, the coccus becomes movable and escapes through an opening in the capsule (ascococcus, from ἀσκός, tube). 2. The coccus divides always in one direction, and some of the divisions remain, like frog-spawn, united by a delicate envelope of glia (*c*, Fig. 21 A); these streptococci are sometimes in motion, wriggling slowly across the field of the microscope, but usually they are at rest; we may find them in fresh secretion from the wound or in pus, and often in alkaline urine, without there being necessarily any bad odor; the streptococcus, along with the isolated micrococcus and gliacoccus, are the forms of coccobacteria which occur most frequently in decomposing secretion from wounds or in diphtheria of wounds. With absolute rest the streptococcus may form long upright filaments, but this is very rare in living organisms, and is difficult to see under the microscope. 3. The coccus grows to rods, which increase in length and then divide across; thus we have bacteria chains (*f*, Fig. 21 A), which may be moving or motionless. In some fluids the division of the bacteria goes on very rapidly, the rods becoming shorter, till they are finally square or rounded; and so between coccus and bacteria there are numerous transition forms. Bacteria are not apt to enter the secretion from wounds, pus, or decomposing blood; on the other hand, they develop and remain in all fluids of the cadaver and in watery exudations of almost all tissues; in the latter they are very movable.

All of these vegetations require plenty of water and organic substances, especially nitrogenous matters, for their rapid propagation; they bear abstraction of water up to a certain point, but if entirely dried out they die; and although they will subsequently swell if placed in water, they have lost their power of vegetating. They can bear temperatures as low as the freezing point, and nearly up to the boiling point; but when it reaches the boiling point they die. In fluids or moist tissues completely excluded from atmospheric air, they will vegetate till all the air contained in the fluid has been used up; then, no more air being absorbed by the fluid, the coccobacteria die, as they cannot cause decomposition of water or any organized combination.

Under these conditions some of these coccobacteria may be thrown into the atmosphere and be generally distributed by the evaporation of fluids, so common in nature. Still, when the air becomes very dry, these vegetations might dry out, die, and become organic but no longer organizable dust; but such an occurrence is provided against. As in many of the algæ of stagnant water, having similar peculiarities and subject to being dried out, so in some of the elements of coccobacteria a larger quantity of peculiarly concentrated protoplasm unites to form a glistening sphere with dark contours, which may be distinguished from other coccus, but hardly from fat globules. These little spheres have the peculiarities of fungus-germs and very resistant seeds; they may be entirely dried, cooled far below the freezing and warmed above the boiling point, and kept hermetically closed for a long time, without losing their germinal activity; hence they are called permanent germs (*Dauersporen*). According to my experience, they form very certainly and not very rarely, under certain circumstances, in bacteria; but they also occur in coccoglia balls; I cannot state whether some spheres of streptococcus also become permanent germs. These *Dauersporen* are the dried germs from which we started for development; they require quiet in or on some fluid or moist body.

I have here given you a brief review of the results to which my investigations on this point have led. But I must call attention to the fact that botanists have not yet proved the correctness of my views, and that they are at variance with those of most others who have investigated this subject, and who consider each of the forms above described as separate plants, and also make numerous species of each kind, especially according to the diseases induced by each. Let me also remind you that most pathologists term these algæ fungi, and often call them all bacteria.

It is to these small organisms that *Pasteur*, and after him *Lister*, attributes decomposition, at least those forms of it whose products are local and general poisons. If we could prevent their entrance into the wound or its secretions, according to this view, there would be no decomposition of the secretions, even if some of them did remain in the wound. With this idea, *Lister* writes a number of rules to be followed in the operation and dressing of the wound, all aiming at the destruction of the germs of coccobacteria which might reach the wound through the hands of the operator and assistants or the air. After the operator and assistants, before each operation or dressing, have carefully washed with soap and water, they dip their hands in a five per cent. solution of carbolic acid; in the same way the parts about the seat of operation are to be carefully washed and moistened with the same solution of carbolic acid; and all instruments, sponges, and dressings used lie in this solution, which is supposed to kill all germs of coccobacteria. To prevent these germs from reaching the wound through the air during the operation and dressing, a two per cent. solution of carbolic acid is constantly sprayed on the part with a special apparatus, so that it falls on the wound in the form of a fine rain. We have already described the occlusion or "antiseptic" dressing, as it is termed, although the open treatment of wounds and some other methods are just as antiseptic. There is no reason for going any further into details here, where we are chiefly explaining principles. *Lister's* dressing, which seems so complicated, is in practice much simpler than would appear from the description; for every step and rule the inventor had a definite reason, and there is nothing arbitrary or intentionally mysterious. If we inquire into the practical working of this treatment, we hear chiefly praise, and many speak enthusiastically of its wonderful effects. Although my own experience with it is not very great, I can recommend it as being generally very good; it is certainly more popular than the open treatment of wounds; it is still a disputed point which of these methods answers best in treatment of wounds of the extremities. I urgently recommend you to perfect yourselves in the principles and practical application of *Lister's* treatment, and you will have many favorable results.

It is different if we accurately examine the correctness of the theoretical views from which *Lister* starts, and inquire whether by his mode of operating and dressing he has attained his object. In regard to the latter point, it has been often proved that in the secretion of wounds treated according to *Lister's* method, and which healed rapidly without reaction, coccobacteria were found about as often as in secretion from wounds which were merely dressed with attention to cleanliness. This shows: 1, that the presence of these vegetations in itself proves nothing about the phlogogenous or other poisonous qualities of the secretion; 2, that *Lister's* dressing is no guarantee for the destruction of bacteria. Against this second point it might be urged that there is no proof that these germs reach the wound *only* from without; it is possible that permanent germs enter the blood through the respired air, and, though they may not develop under normal circumstances, do so in the secretion of wounds. If this be possible, there is no sense in the theory of *Lister's* method as far as regards its attacking organic germs by chemical means. Indeed, it is my opinion that those not very frequent cases where coccobacteria vegetations have been found in completely closed, deeply-seated points of inflammation, which never communicated with the air, can only be explained in the way above mentioned. Apart from the fact that *Lister's* dressing is expensive if followed out in all its details, and that more or less severe poisoning is often caused by the annoying dermatitis induced by the carbolic acid, this incongruence of theory and practice has led to the employment of more and more dilute solutions of carbolic acid, and its replacement by other antiseptic and less irritating acids and salts (salicylic acid, *Thiersch;* sulphide of sodium, *Minich*). Various changes have also been made in the mode of applying the dressing (*Volkmann, Bardeleben*); the spray has been entirely omitted, and in its place after the operation the wound has been washed with a more concentrated antiseptic solution, etc., etc. Thus *Lister's* dressing has been variously modified, and from each modification the same favorable results have been obtained as from the original dressing. This confirms me in the opinion formed when this method was first described, and which I have already stated, that the scrupulous cleanliness and the careful removal of secretion from the wound is the most important part of it, and that it is chiefly popular among surgeons who formerly paid less attention to these points, and left the dressings to the dirty hands of nurses or to careless students or young physicians, while now the dressings are all applied according to definite principles of cleanliness. Moreover, the constantly spreading and more energetically preached doctrine of local infection from wounds, of which we shall treat hereafter, has led to a recognition of the necessity of a rational treatment of wounds, and has contributed essentially to opening the way for the open treatment of wounds—to *Lister's* method and antiseptic lotions.

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There is still another mode of adhesion of the edges of wounds, which consists in the direct union of two adjacent granulating surfaces. This mode of healing, which you may call healing by the third intention, is unfortunately very rare. The reason of this is evident: pus is constantly secreted from the surface of the granulations, and while this goes on the surfaces are only apparently in contact, for there is pus between them. Occasionally, it is true, we may, by pressing the two granulation surfaces together, prevent the further formation of pus, and then the two surfaces may adhere; we accomplish this by drawing the flaps of the wound firmly together with good adhesive plaster, or by the application of secondary sutures, for which it is well to employ wire. Unfortunately, the attempt to hasten the cure by these means so rarely succeeds, that they are only exceptionally employed. The best results are obtained from secondary sutures when, six or seven days after the injury, they are applied about four or five lines from the edge of the wound, because the tissue is then more dense and firm, and the sutures cut through less quickly.

There is still another mode of healing, viz., healing of a superficial wound under a scab. This only occurs frequently in small wounds, that secrete but little pus, for in these alone does the pus dry on the wound to a firmly-attached scab; in profuse suppuration it is true the superficial layer of the pus may dry by evaporation of the watery portion, but, while new pus is constantly being secreted under it, it cannot form an adherent, consistent scab. When such a scab has formed, the granulation tissue develops to only a very small amount under it; perhaps because on account of the slight pressure of the scab, the granulation tissue is less mucous, so that the epidermis can more readily regenerate under the scab; such a small wound may be wholly cicatrized when the scab falls.

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The granulation surface may assume a totally different appearance from that above described, especially in large wounds. There are certain *diseases of the granulations*, whose marked forms I shall briefly sketch for you, although the varieties are so numerous that you will only learn them from individual observation. We may divide granulation surfaces as follows:

1. Proliferating fungous granulations. The expression "fungous" means nothing more than "spongy;" hence by fungous granulations we mean those that rise above the level of the skin, and lie over the edges of the wound, like fungus or sponge. They are usually very soft; the pus secreted is mucous, glairy, tenacious; it contains fewer cells than good pus, and most of the pus-cells, like granulation-cells, are filled with numerous fat-globules and mucous material, which is also more abundant than normal as intercellular substance; and in these granulations *Rindfleisch* also discovered collections of *Virchow's* mucous tissue, fully developed. The development of vessels may be very prolific; the fragile tissue often bleeds on the slightest touch; occasionally the granulations are of a very dark blue. In other cases the development of vessels is very scanty, often to such a degree that the surface is light red, or in spots has even a yellowish, gelatinous appearance, in very anæmic persons, often also in young children and very old persons. The most frequent cause of development of such proliferating granulations is any local impediment to the healing of the wound, such as rigidity of the surrounding skin, so that the con

traction of the cicatrix is difficult; a foreign body at the bottom of a tubular granulating wound (a fistula); this abnormal proliferation is also particularly apt to occur in large wounds, which can only contract slowly; it appears as if the activity of the tissue was occasionally exhausted, and no longer capable of continuing the requisite condensation and cicatrization, so that it only produces relaxed, spongy granulations. As long as there are granulations of the above character, rising above the edges of the skin, cicatrization does not usually progress. The wound would probably heal, but not for a very long time. We have plenty of remedies for hastening the healing under such circumstances; these are especially caustics, by which we partly destroy the granulation surface, and thus excite a stronger growth from the depth. At first you may cauterize the granulating surface daily, especially along the edges, with nitrate of silver, whereupon a white slough will quickly form, which will become detached in twelve to twenty-four hours, or even sooner; repeat this cauterization as required, till the granulating surface is even. Another very good remedy is sprinkling the wound with powdered red precipitate of mercury (hydrar. oxyd. rubrum), which also should be repeated daily, to improve the granulating surface. Compression with adhesive plasters also acts very well occasionally. If the granulations be exceedingly dense and large, we often may succeed soonest by cutting some of them off with the scissors; the consequent hæmorrhage is readily arrested by applying charpie. Where the proliferation is less, astringent lotions, such as decoction of oak-bark, cinchona-bark, lead-water, etc., may answer to excite the sluggish cicatrization.

2. By *erethitic granulations* we mean those characterized by great pain on the slightest provocation; they are usually very proliferant granulations, which readily bleed; it is a very rare condition. In excessive erethism of the granulations, they are so sensitive that they cannot endure the slightest touch or any dressing; a less degree of sensitiveness of the granulations is not so rare. On what it depends, is not very certain; granulation tissue itself has no nerves; in most cases touching it causes no sensation, only the conduction of the pressure to the subjacent nerves causes sensation. In the above excessive sensibility, probably the ends of the nerves at the floor of the wound are degenerated in a peculiar manner; perhaps there are miniature thickenings of the finest nerve-ends, like those that we shall hereafter see on large nerve-trunks. It would be a thankworthy task to make a careful examination of this question. We occasionally observe similar conditions in the cicatrices in large nerves, and shall speak of this hereafter. For this very painful sensitiveness, which not only interferes with healing, but greatly worries the patient, you may first try soothing

ointments, almond-oil, spermaceti-ointment, or simple cataplasms of boiled oatmeal or linseed-meal, or warm-water compresses. The narcotic compresses or cataplasms, made with the addition of belladonna or hyoscyamus-leaves, are of little benefit. If these applications do not answer, do not delay destroying the entire granulating surface, or at least the painful part, with caustic (nitrate of silver, caustic potash, or the hot iron), with the patient anæsthetized, or else excising the entire surface with the knife. If the great painfulness be due to hysteria, anæmia, etc., you will not attain much by any local remedies, but should try to assuage the general irritability by internal remedies, such as valerian, assafœtida, iron, quinine, warm baths, etc.

3. In large wounds, especially in fistula granulations, a yellow rind sometimes forms on part of the granulation surface, which may be readily detached, and on careful examination is found to consist of pus cells, very firmly attached together. Although I have sometimes found coagulating filaments between the cells, they do not always occur, hence we must suppose that the cell-body, the protoplasm itself, is transformed into fibrine, as occurs in true croup, and especially in the formation of fibrinous deposits on serous membranes. Here there is also a *croup of the granulations.* The croupous membrane reforms even a few hours after its removal, and this is repeated for several days, till it either disappears spontaneously, or finally ceases on cauterization of the affected part. Very similar white spots are occasionally found on larger granulation surfaces, which are probably not caused by fibrinous deposits, but by local obstruction of the blood-vessels. Under peculiar, unfavorable conditions, both states may result in destruction of the granulations, in a true *diphtheria* of the wound, which we shall hereafter treat of as *hospital gangrene.* Fortunately, however, it rarely goes on to this disease, but the state of the wound improves again after a time, and the recovery takes the usual course.

If disease of the granulating surface be accompanied by swelling, great pain, and fever, we have a true acute inflammation of the wound; then the mucous granulation substance sometimes coagulates throughout to a fibrinous mass; the wound-surface looks yellow and greasy. I shall treat of the causes of these secondary inflammations under the head of contused wounds. Usually the croupous inflammation, which has affected part or the entire surface of a wound, ends in sloughing of the diseased granulations, whereupon new granulations spring from the depths.

It cannot be denied that the perfectly local, superficial, and interstitial deposit of fibrine strongly supports the view that *Virchow* has proposed for croupous processes generally. It was formerly sup posed that in all inflammatory croupous process, especially in the ordinary form of acute inflammation of the lungs and pleura, the blood was over-rich in fibrine; that there was a fibrinous crasis in the blood, as a result of which, the excessive fibrine escaping from the capillaries, coagulates partly on, partly in, the inflamed surface, and so led to the formation of these pseudomembranous deposits. *Virchow*, on the other hand, proposed the idea that, by the inflammatory process, the tissue may be placed in a condition to cause coagulation of the fibrinous solution infiltrating it. I cannot here enter more particularly into the various grounds on which *Virchow* bases this view, but shall only call attention to the fact that in the case in question (of fibrinous exudation on the granulating surface), at least there can be no rapidly coming and evanescent fibrous crasis of the blood; but evidently it is a local process which may readily be removed by local remedies. According to the repeatedly-mentioned observations of *A. Schmidt*, we may infer that in certain quantitative and qualitative irritations of the tissue, more fibrogenous tissue than usual escapes from the capillaries. *Virchow* had even previously called attention to the fact that, from repeated irritation, simple serous exudation may become fibrinous or croupous. If you apply a spanish-fly blister to the skin, a vesicle filled with serous fluid forms—the superficial layer being lifted from the rete mucosum by the rapidly-forming serous exudation; if we remove the vesicle and reapply the blister, in many cases after a few hours we shall find the surface covered with a fibrinous layer, which contains innumerable newly-formed cells; indeed, is almost entirely composed of them. We may attain the same result by applying the plaster to skin already inflamed, or to a young cicatrix.

The treatment of croupous inflammation of the granulations is purely local; we should carefully seek for any causes of new irritation, and try to remove them. Daily remove the fibrinous rinds, and cauterize the exposed surface with nitrate of silver, or paint it with tincture of iodine, and you will soon see this abnormal state of the granulating surface disappear.

4. Besides the above diseases of the granulations, there is occasionally a state of perfect relaxation and collapse, in which they present an even, red, smooth, shiny surface, from which the nodular, granular appearance has entirely disappeared, and, instead of pus, a thin watery serum is secreted. This state almost always occurs in the granulations at the end of life; as already mentioned, you always find it in the cadaver.

It is still necessary to add something about the cicatrices, concerning certain subsequent changes in them, their proliferation and their shape in different tissues.

Linear cicatrices of wounds, that have healed by first intention, rarely undergo subsequent degeneration. Large, broad cicatrices, especially when they lie immediately on the bone, often open again; the epidermis, which is tender at first, being torn off by motion or by the least blow or friction, and there is superficial atrophy, an excoriation of the cicatrix. Sometimes the young epidermis is elevated like a vesicle, by exudation from the vessels of the cicatrix; there may also be some hæmorrhage, so that the vesicle will be filled with bloody serum. Then, after removing the vesicle, you have an excoriation, as after simple rubbing off of epidermis. This opening of the cicatrix, if often repeated, may prove very annoying to the patient. You prevent this most readily by causing the patient to protect the young cicatrix for a time with wadding or a bandage. If the excoriation has taken place, apply only mild dressings: oil, glycerine, zinc-salve, etc., or emplastrum cerussa. In these cases, irritating salves enlarge the wound, and consequently should be avoided.

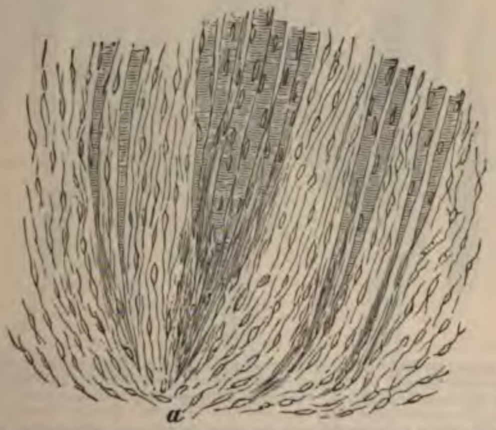
If the granulating surface be once perfectly covered with epidermis, as already stated, the retrogressive changes to solid connective tissue take place in the cicatrix, and it atrophies. But in rare cases the cicatrix grows independently, and develops to a firm connective-tissue tumor. This is seen almost exclusively in small wounds that have long suppurated and been covered with spongy granulations, over which the epidermis formed exceptionally. You know it is the custom to pierce the ear-lobes of little girls, so that they may subsequently wear ear-rings. This little operation is done with a coarse needle by the mother or the jeweller, and a small ear-ring is at once introduced through the fresh puncture. As a rule, this puncture soon heals—the ring preventing the closure of the opening. But in other cases there are active inflammation and suppuration; indeed, if the suppuration continue, the ring may cut downward through the lobe; granulations develop at the openings of entrance and exit; finally, the trial is given up, and the ring removed; then the opening often heals quickly. In other cases the granulations cicatrize, the cicatrix continues to grow, and on both sides of the lobe of the ear small connective-tissue tumors, small fibroids, form. These look like a thick shirt-button drawn through the hole of the ear, and they grow independently like a tumor. If you examine these tumors, on section you find them of pure white tendinous appearance, like the cicatrix itself. Microscopically the tissue is found to consist of connective tissue with numerous cells; it is simply a proliferation, an hypertrophy of the cicatrix. I have seen this twice in the ear; another case is mentioned by *Dieffenbach* in his operative surgery. I once saw similar tumors on the back of the neck, where they had formed at the openings made for a seton; they were about the size of a horse-chestnut. They should be carefully removed with the knife, and any subsequent granulations kept in subjection by nitrate of silver.

[The translator has seen the above tumors on the lobe of the ear several times; in all but two instances they occurred in mulatto females; in one case the tumor had returned after a previous removal.]

In the above description of the formation of granulations and cicatrices, for the sake of simplicity we have only referred to the process as it is found in connective tissue, but must now speak of it as it occurs in cicatrization of other tissues.

The cicatrix in muscle is at first almost entirely connective tissue; in the ends of the muscular fibres there is at first destruction, then at a certain boundary a collection of nuclei; then there is rounding off of the fibres, sometimes club-shaped, sometimes of more conical form, and the stumps of the muscular fibres unite with the connective tissue of the cicatrix just as they do with the tendons; the muscle cicatrix becomes an inscriptio tendinea. I myself have only observed them in wounds of muscle that had healed by first intention, and have never there seen any thing that I could decide was a new formation of muscular tissue. In suppurating ends of muscle, *O. Weber* has witnessed a slight formation of new muscle; this appears to occur chiefly in formation of granulations on muscle and in certain tumors.

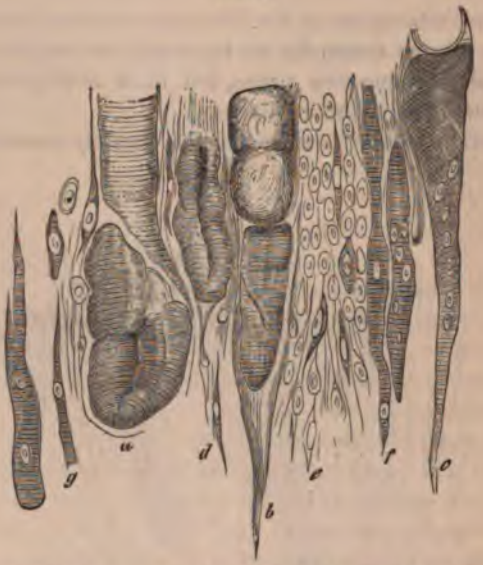
FIG. 18.



Cicatrix from the upper lip of a dog. *a*, connective tissue of the cicatrix. The divided muscular fibres are here atrophied for a short distance, and terminate in a conical shape. Magnified 300 diameters.

*Weber* is of the opinion that young muscular fibres typically form from the cells of old ones, but considers it impossible to prove that no muscular cells originate from other young cells. As a result of his examination of old muscular cicatrices, he also maintains that the regeneration continues a long time, and in most cases is more complete than is generally supposed. *Maslowsky* has affirmed the metamorphosis of wandering cells to muscle-cells; but I consider the *cinnabar method* employed by him as insufficient to prove this assertion. [Cinnabar or vermilion injected into the blood is taken up by white corpuscles, and may afterward be discovered on inflamed tissue.]

FIG. 19.

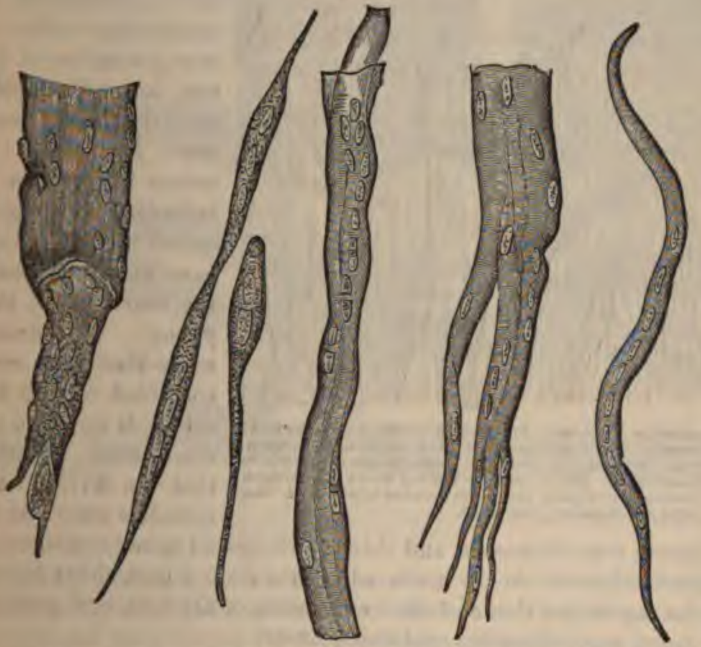


Ends of divided muscular fibres from the biceps muscle of a rabbit eight days after the injury; *a b c*, old muscular fibres; *a*, the contractile substance rolled up and balled together; the same way in the bundle above *d*; the same with the sarcolemma drawn out to a point; *c*, into the pointed cornet-shaped sarcolemma tube extends a series of young muscular nuclei, between which there is very delicate transversly striated substance; *e*, the same with young, free muscle-cells; *f*, two young ribbon-like muscular filaments; *g*, the same of various size isolated Magnified 450 diameters; after *O. Weber.*

*Gussenbauer* has shown that, after injury, the muscular filaments usually break down into flakes, and then new young muscle-cells form, after the type of embryonal development, from the cells contained in the old muscle-filaments; the amount of the new formation depends on the quality and duration of the irritation.

If a *nerve* be divided, its ends separate, from their elasticity, they swell slightly, and subsequently unite by development of a new formation of true nerve-tissue, so that the nerve is again capable of conduction through the cicatrix. In large superficial cicatrices, new nerves develop; when you have excised portions of skin and have brought together and united parts lying at a distance, new nerves grow through the cicatrix and perfect power of conduction comes after a time, as may be often observed in plastic operations. These facts are very remarkable, and physiologically are still entirely inexplicable. Just think how wonderful that these nerve-filaments, sensory and motor, should find each other in the new adhesion, and that even, as we must suppose, the stumps of the primitive fibres should unite as they had been united, so that correct conduction and localization might result as they actually do! We cannot here go more exactly into this subject. I will only mention that the more minute process, which has been very carefully followed by *Schiff*, *Hjelt*, and others, is generally as follows: first, in the stump of the nerve there is a destruction of the nerve-sheath, possibly also of the axis cylinder to a certain extent; at the same time in the neurilemma there is a collection of cells, which proceeds to the development of spindle-shaped cells in the substance lying between the ends of the nerve, and extending into the

FIG. 20.



Regenerative processes in transversely-striated muscular fibres after injury. Magnified about 500, after *Gussenbauer*.

stump. From these cells, just as in the embryo during intra-uterine life, new nerve-fibrillæ develop upward and downward; the filaments, which are at first very pale, subsequently acquire a sheath, and then cannot be distinguished from ordinary nerve-filaments.

FIG. 21.

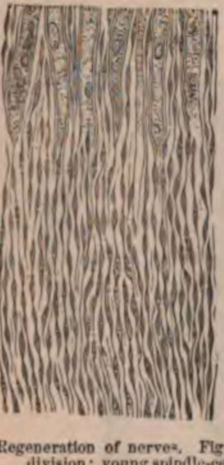
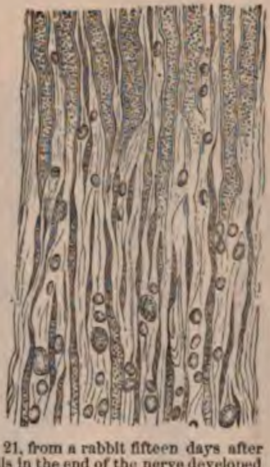


FIG. 22.



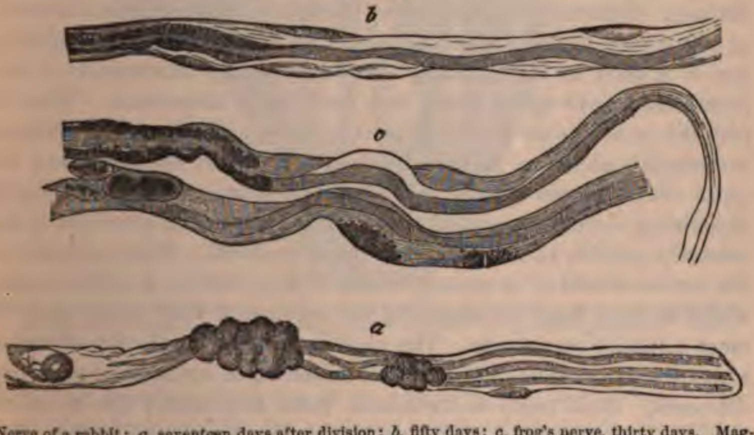
Regeneration of nerves. Fig. 21, from a rabbit fifteen days after division; young spindle-cells in the end of the nerve developed from the connective tissue and intimately connected with the neurilemma. Fig. 22, from the frog ten weeks after division; development of young nerve-cells from the spindle-cells. Magnified 300 diameters, after *Hjelt*.

The most recent investigations as to the significance of wandering cells in new formation of tissue, as well as the special studies over the formation of nerves in portions of tadpoles' tails regenerated after injury, have made me doubt the former view, that young regenerated nerve-filaments were composed of spindle-cells. It seems to me much more probable that the divided axis-cylinders grow out into young nerve-filaments, and that the elongated spindle-cells, which undoubtedly exist in the nerve-callus in certain stages, either belong to the connective tissue of the neurilemma or are detached portions of young nerve-filaments containing nuclei.

The last investigations of *Neumann* and *Eichhorst* confirm previous ones in regard to the immediate results of division, but show that the young nerve-filaments grow directly from the axis-cylinder, as well from the central as from the distal part, meet together, and blend, as the offshoots from a capillary wall sink into the wall of another vessel, and so may form a communicating canal between two vessels (*Arnold*). The process in the wounded nerve corresponds most beautifully with that in wounded muscle. In the muscular as in the nervous filament several young filaments sprout fron. one primitive filament (*a*, Fig. 22 A; compare Fig. 20).

So it is shown that muscles, vessels, nerves, and epithelium are not regenerated from proliferating connective-tissue cells or wandering cells, but from throwing out offshoots from their tissue, or from cells derived from the protoplasm of their tissue. It is very probable that connective-tissue cells also, especially those still containing protoplasm, send out offshoots at the wounded part in which nuclei subsequently form, as is done in the nerves of the tadpole's tail. This point should be investigated again; till then we may regard wandering cells as the source of the young regenerated tissue. Since *Schwann's* teaching about the development of tissue from cells, we are so convinced that every new tissue proceeds from young cells, that the announcement of independent growth of a perfect tissue without intervention of cells finds little credit; and the increase of cells by offshoots, with subsequent development of nuclei in these offshoots, is a procedure that histologists have long kept in the background, substituting for it cell-division, although botanists have ascribed a very prominent rôle to this mode of development in plants. From the latest published observations we see that the capillary walls, the axis-cylinder of nerves, and the contents of muscular filaments possess this capacity for outgrowth without direct participation of new cells. *Rokitansky* ascribed to connective tissue the capacity for independent outgrowth.

FIG. 22 A.

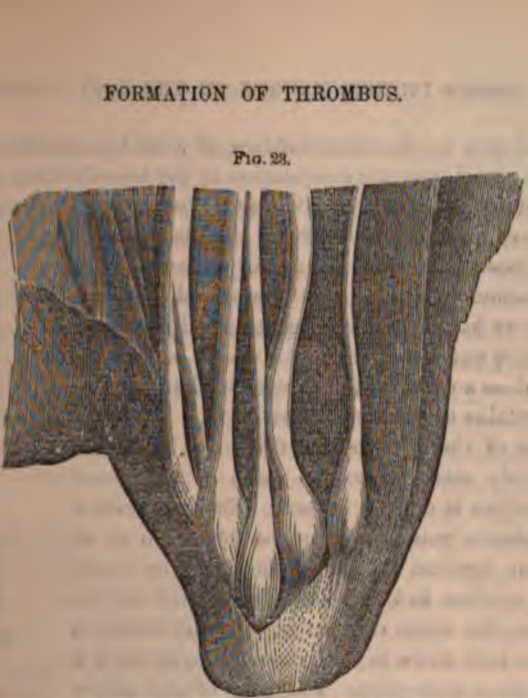


Nerve of a rabbit: *a*, seventeen days after division; *b*. fifty days; *c*, frog's nerve, thirty days. Magnified about 600. After *Eichhorst*.

In the human being the regeneration of nerves only takes place within certain limits, which, it is true, cannot be very accurately defined. The complete regeneration of large nerve-trunks, as of the sciatic or median nerves, does not occur; nor does it take place after excision of large portions of nerve, if the ends remain, say three or four lines apart. Very accurate apposition of the ends of the nerve is necessary, for apparently the transformation of the newly-formed intermediate substance to nerve-substance can only take place by means of the nerve-stump, although there are different opinions about the mode of this process; we shall see similar conditions in the healing of broken bones, where bony union only follows accurate coaptation of the fragments. Now, how is it in this respect with brain and spinal tissue? In the human being there is no regeneration here after injury, or after loss of substance from idiopathic inflammation, or at least not sufficient to restore the power of conduction. In animals, indeed, as *Brown-Séquard* has shown in pigeons, after dividing the spinal marrow, there may be regeneration with disappearance of the paralysis, which has of course occurred in all parts below the point of division. Unfortunately, this power of regeneration of nerves decreases in proportion to the higher development of the vertebrate animals, and it is least in man. As is known, in young salamanders whole extremities grow again when they have been amputated. What a pity this is not so in man! However, as regards the nerves, Nature occasionally seems to make a fruitless attempt at regeneration; for quite often the nerve-ends in amputation-stumps, instead of simply cicatrizing, develop to club-shaped nodules, which are occasionally excessively painful, and require subsequent excision. These nodules on the nerves consist of an entanglement of the primitive nerve-filaments, which develop from the stump of the nerve as if they would grow to meet opposite nerve-ends. The cicatrices in the continuity of nerves also are sometimes nodular from the formation of convoluted primitive filaments. Such small nerve-tumors (true neuromata) are occasionally excessively painful, and must be removed with the knife. But there are also traumatic neuromata, which are not at all painful, as I have seen in old amputation-stumps. In general, these proliferations of nerve-cicatrices are to be compared with the previously-mentioned hypertrophy of connective-tissue cicatrices, and with proliferating bone, which, although rarely, is formed in great excess in the healing of broken bones.

The process of healing after injury of great vessels, especially of arterial trunks, has been carefully determined by experiment. If a large artery be ligated in an amputation or for disease in its continuity, as the ligature is drawn tight, the tunica intima is ruptured, and the tunica muscularis and adventitia are so constricted that their inner surfaces folded up lie in exact apposition. You may satisfy yourselves of the frequent although not necessarily universal rupture of the internal tunic, by ligating a large arterial trunk in the cadaver, for you not unfrequently experience a slight grating or crackling under the finger when tightening the ligature; you may also see it on cutting open a ligated artery after detachment of the ligature. From the point of ligation to the next branch leaving the artery, both at the central and peripheral ends, the calibre of the vessel fills with coagulated

FIG. 23.

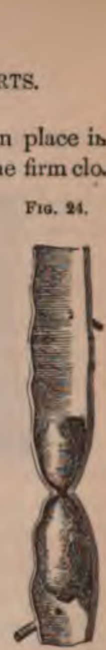


Nodular nerve-terminations in an old amputation-stump of the arm. From a preparation in the Anatomical Museum at Bonn. Copied after *Froriep*, "Surgical Copperplates." Bd. I., Taf. 113.

blood, the so-called *thrombus* (from ὁ ϑρομβος, the blood-clot). The enveloping ligature kills the enclosed tissue, which gradually breaks down into pus, and when this process is completed the ligature falls, or, as we technically express it, "the ligature has cut through," "comes away." When this has taken place, the calibre of the artery must be permanently and certainly closed, or there will at once be another hæmorrhage. Under unfavorable circumstances it may certainly happen, in small as well as in arteries of medium or large size, that the ligature cuts through too soon, and then dangerous, sudden secondary hæmorrhage occurs. We may foresee this if the wall of the artery was diseased; often calcified arteries cannot be ligated, as the ligature does not compress them or cuts through them at once; sometimes the artery is softened (as, for instance, when part of its course has been through the wall of a large abscess) so that on ligation the ligature cuts through and must be applied farther up. But unfortunately, in perfectly healthy subjects, as I found in the last war, hæmorrhages too often occur from the point of ligation of large arteries, where carefully-applied ligatures cut through before the organic closure was firm enough to resist the current of blood; this greatly impairs the value of such operations, which are often temporarily necessary to save the patient's life.

Passing now to the consideration of what has taken place in the end of the vessel from the coagulation of the blood till the firm closure, experiments on animals and accidental observations on man have given the following: the blood-clot at first lying loose in the vessel gradually becomes more firmly attached to the wall of the vessel, and constantly grows harder, but still remains red for a long time; it does not lose its color for weeks or months, and then does so first in the centre, so that the rest of it still retains a slight yellowish tinge. After the detachment of the ligature, the thrombus is so hard and so firmly attached to the walls of the vessel that the calibre is entirely closed. The preparation (Fig. 24) shows you the thrombus formation in an artery after ligation in the continuity; the lower thrombus reaches to the point of departure of the first branch, the upper one not so far; the former is the rule as laid down in most books, the latter is a not uncommon exception. Plugging of the artery by a blood-clot, which becomes firm, is, however, only a provisional state, for the thrombus does not remain so for all future time, but the cicatricial tissue shrinks and atrophies; this takes place in the course of months and years, at which time the closure of the artery at the point of division has become solid by adhesion of the walls of the vessel. If you examine such an artery a few months after the ligation, you find nothing of the thrombus; but the artery terminates in a conical point of cicatricial connective tissue.

FIG. 24.

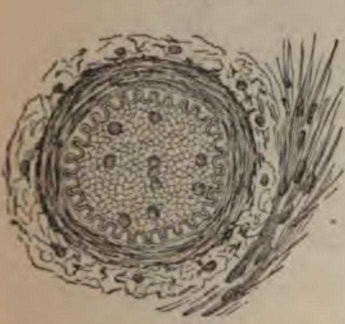


Artery ligated in the continuity. Thrombus; after *Froriep.*

The above changes, which we may follow with the naked eye, show that in the blood-clot there is a change which essentially consists in its increasing firmness and coherence to the wall of the vessel; we shall now study with the microscope on what this transformation of the blood-clot depends. If you examine the recent blood-clot, you find it to consist of red blood-corpuscles, a few colorless blood-cells, and of fine filaments and coagulated fibrine, arranged in irregular network. If you take a thrombus two days after the ligation of a small or medium-sized artery, it is firmer than at first, and is broken up with difficulty; the red blood-cells are little changed, the white ones are greatly increased; they have sometimes two and three nuclei as previously, sometimes single pale, oval nuclei with nucleoli; some of these cells are almost double the size of white blood-cells. The fine filaments of the fibrine are united to an almost homogeneous mass, which is difficult of division. If you again examine a thrombus six days old, the red blood-cells have almost disappeared, the fibrine is more firm and homogeneous, and even more difficult to separate than previously; a large number of spindle-shaped cells with oval nuclei, showing distinct divisions, appear. From the above, it appears that even quite early a number of living cells appear in the blood-clot, whose further development will be seen from what follows. Since we obtain a more accurate understanding of the changes in the thrombus and its relation to the arterial walls, by making transverse sections of the thrombosed artery, we shall proceed to do this.

This preparation shows a transverse section of a recent thrombus in a small artery; within, the delicate mosaic formed by the crowded red blood-corpuscles, among them a few round white blood-cells (which have been rendered visible by carmine); next comes the tunica intima, laid together in regular folds, in which the blood-clot clings; then the tunica muscularis; then the tunica adventitia, with the net-work of elastic fibres; to the right some adherent loose connective tissue. The next preparation (Fig. 26) is the transverse section of a human artery, closed with a thrombus for six days; we see no red blood-cells; the white ones are greatly increased, mostly round; but, in the tunica adventitia and surrounding connective tissue, there has already been some cell infiltration. If we now examine a ten-day-old thrombus from a large muscular artery of the thigh of a man (Fig. 27, *a*), we find it already containing numerous spindle-cells, which are partly arranged in striæ (subsequently vessels); the intercellular substance is filamentary, here rendered transparent by acetic acid. Finally, there is also formation of blood-vessels in the organized thrombus, as you see in the following preparations (Figs. 28 and 29).

Fig. 25.

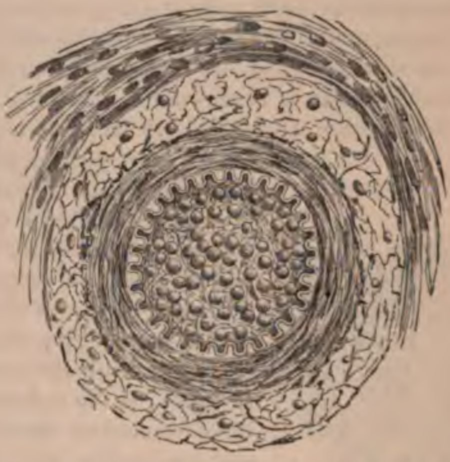


Transverse section of a fresh thrombus. Magnified 300 diameters.

It has been established, by the investigations of *O. Weber*, that the vessels of the thrombus communicate partly with the calibre of the thrombosed vessel, partly with its vasa vasorum.

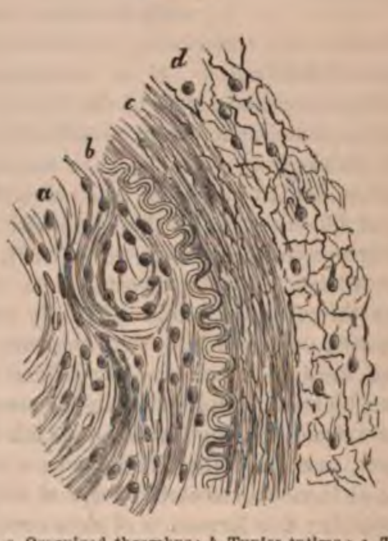
The process of healing in transversely-divided veins appears at the first glance to be much simpler than in the arteries; even in the large veins of the extremities, the divided ends fall together, and appear to heal at once, as soon as the blood has been obstructed at the next valve above; at these valves clots form, and they are often much larger than is desirable; this formation of clots extending toward the heart will hereafter occupy our earnest attention. But I have of late observed that the tunica intima of the divided vein does not by any means so fold together and adhere, but that here also there is a clot, although a small one, which is organized like the arterial thrombus.

FIG. 26.



Transverse section of a thrombus six days old. 300 diameters.

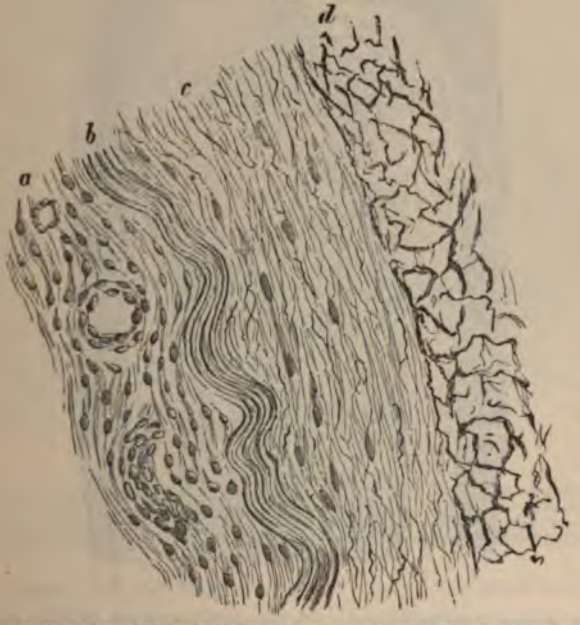
FIG. 27.



Ten-day-old thrombus. *a*, Organized thrombus; *b*, Tunica intima; *c*, Tunica muscularis; Tunica adventitia. 300 diameters.

If you draw conclusions from these preparations, presented in such a fragmentary way, it appears that in the clotted blood there is a cellular infiltration, which here leads to development of connective tissue; in short, that the thrombus becomes organized. The thrombus is not a permanent tissue, but gradually disappears again, or, at least, is reduced to a minimum, a fate which it shares with many new formations resulting from inflammation.

Fig. 28.

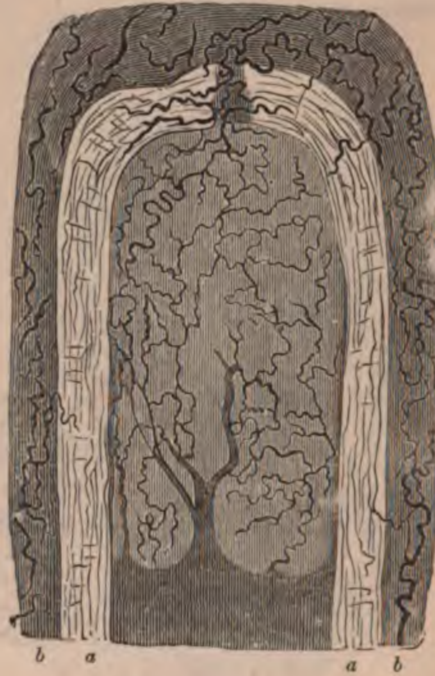


Completely-organized thrombus in the human arteria tibialis postica. *a*, Thrombus with vessels, perfectly united with the innermost layer of the intima; *b*, the lamellæ of the tunica intima; *c*, the tunica muscularis, traversed by numerous connective tissue and elastic filaments; *d*, Tunica adventitia. Magnified 300 diameters. After *Rindfleisch*.

Peculiar reasons caused me to investigate more accurately the organization of the thrombus. The importance of this process is rather extensive; a point on which you cannot at present judge well, but will hereafter be in a position to estimate fully, when we come to treat of diseases of the vessels.

From my investigations up to the present time, I do not think I dare retract the assertion that coagulated fibrine *may*, by aid of cells, be transformed into connective-tissue intercellular substance, although I cannot decide whether this be due to true metamorphosis, or to a gradual substitution of cell protoplasm for disappearing fibrine. Some have attempted to refer the origin of the cells, which appear in constantly-increasing numbers in the thrombus, to the wall of the vessel; the arteries, as well as the veins, are coated with a lining of epithelium, which to some extent represents the innermost lamella of the tunica intima. These epithelial cells and the nuclei of the striated

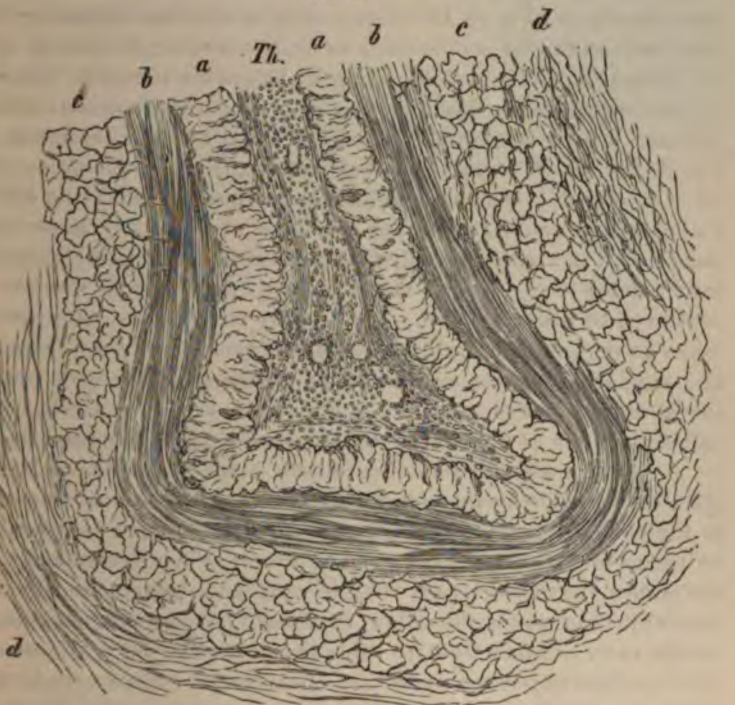
FIG. 29.



Longitudinal section of the ligated end of the crural artery of a dog, fifty days after ligation the thrombus is injected; *a a*, tunica intima and media; *b b*, tunica adventitia. Magnifie 40 diameters.

lamellæ of the intima have been claimed *a priori* by some authors, so that they could let new cells be formed from them, and grow into the thrombus; in his last work, *Thiersch* also inclines to this view. I acknowledge that I myself formerly strongly combated the supposition that the blood could of itself become organized to connective tissue with vessels; but from examinations of transverse sections of thrombosed arteries, I am satisfied of its correctness. After having abandoned the idea of proliferation of stable tissue-cells in inflammation, we can no longer talk of a proliferation of the intima in the old sense. But whence come, then, these newly-formed cells? I have no doubt that they originate from the white blood-cells, which have been

Fig. 30.



Portion of a transverse section of a human femoral vein, with an organized vascular thrombus, 18 days after amputation of the thigh; *a a*, Tunica intima; *b b*, media; *c c*, adventitia; *d d*, enveloping cellular tissue; *Th*, organized thrombus with vessels; the layering of the fibrine is still distinctly visible in the periphery of the thrombus. Magnified 100 diameters.

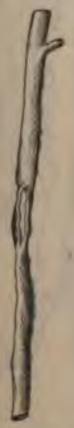
partly enclosed in the thrombus, partly may have wandered into it, according to the observations of *V. Recklinghausen* and *Bubnoff*. As regards the red blood-cells, it seems that they gradually unite with the coagulated fibrine, lose their shape, become intercellular substance, and lose their coloring matter, which is separated as granules or crystals of hematoidin. Little as we know whence blood-cells come, and whither they go, still it is certain that the white cells enter the blood from the lymphatic vessels, and that they enter the latter from the lymphatic glands or connective tissue elsewhere; they are cells that originate directly from connective-tissue cells, or from a protoplasm analogous to connective tissue. Are these cells still viable when enclosed in a blood-clot? After coming to rest here, can they transform themselves to tissue? It is impossible to affirm or deny these questions absolutely; since *Bubnoff* has shown that wandering cells enter the thrombus, and may there continue their movements, there is no necessity for supposing that the white blood-cells (which are identical with wandering cells) enclosed in the thrombus, on coagulation, no longer move, and cannot be transformed into tissue. Hitherto there have been no investigations as to whether wandering cells pass through the walls of arteries as readily as through those of veins, as *Bubnoff*'s investigations only refer to venous thrombi. Some of my investigations in this direction showed me that minute cinnabar granules passed through the carotid of a dog into the thrombus, but I could not satisfy myself that they were replaced by wandering cells. So at present it is uncertain whence the numerous wandering cells in an organizing arterial thrombus originate, and how they enter there. *Tschausoff*, in a very carefully-studied work that has lately appeared, calls attention to the fact that a great portion of large thrombi are destroyed by disintegration. This is very true, but he goes too far when he entirely denies the provisional organization of the thrombus, and supposes that the disintegration of the clot is immediately followed by the adhesion of the walls of the vessel, to which I have called attention as the definite termination of the whole process.

As I have already stated, peculiarly favorable conditions are requisite for the blood-clot to become organized. It is an absolute law in the human organism, that non-vascular tissues, which are nourished by means of cells alone, have no great extent; the articular cartilages, the cornea, the tunica intima of these vessels, the tissues, are all in thin layers; in other words, the cells of the human body cannot, like those of plants, carry nutrient fluid to any given distance, but are limited in their conductive power; at certain distances new blood-vessels must appear, to supply and carry off the nutrient fluid. The blood-clot, consisting of cells with coagulated fibrine, is at first a non-vascular cellular tissue, which can only maintain its existence in thin layers. This appears from observations, which we shall hereafter often have occasion to mention; namely, that large blood-clots are not organized at all, or only in their peripheral layers, while they disintegrate in the centre. From this it appears that, in healing by the first intention, a small amount of blood lying between the edges of the wound does no harm, while a larger amount interferes with healing, or prevents it altogether. You will soon be able to verify this observation in the clinic.

The formation and organization of the thrombus have engaged the attention of surgeons and anatomists since the time of *John Hunter* and even yet they are not fully understood. We must consider them here on account of their general histogenetic interest, although of late it is doubtful whether thrombi are practically as important for the results of ligation as was formerly supposed. Even *Porta* called attention to the fact that the quick adhesion and union of the tissue around the ligated artery was as important as organization of the thrombus. Surgeons have kept this point well in view always striving, by most carefully operating and attending to the wound, to attain healing by the first intention. But it was the success of acupressure which first showed clearly that the adhesion of the tissues by coagulable exudation even in forty-eight hours is enough to keep securely the compressed or twisted artery, even when it is the size of the femoral. Although *Kocher* has shown that, even after acupressure, thrombi occur in arteries, yet they are too small to check bleeding in a large artery within forty-eight hours. Hence, even from this point of view, attempts to replace the ligature by other methods, which leave no threads in the wound but permit its entire closure by first intention, should be encouraged without denying in any way the extraordinary advantages of the ligature.

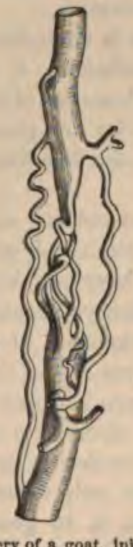
Let us now look at the fate of the circulation after ligating a large artery in the continuity. Suppose that, for a hæmorrhage in the leg, the femoral artery has been ligated; how does the blood now reach the leg? how will the circulation go on? Just as on closure of capillary districts, under increased pressure, the blood presses through the next permeable vessels, which are thereby dilated; the same thing occurs on closure of small or medium-sized arteries. Under increased pressure, the blood flows through the branches close above the thrombus, and from the numerous arterial anastomoses, both in the

FIG. 31.



Carotid artery of a rabbit, injected 6 weeks after ligation. After *Porta*

FIG. 32.

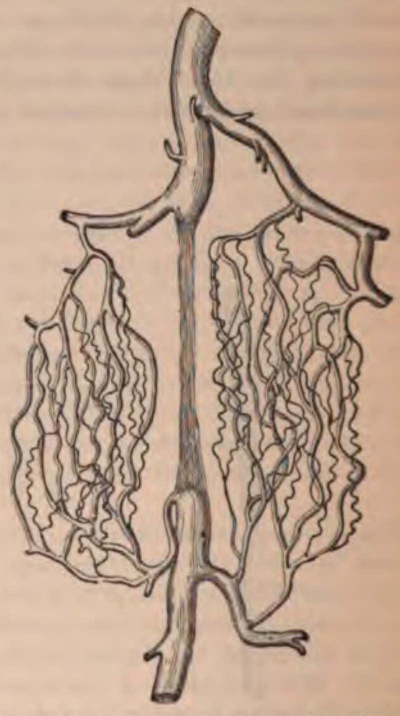


Carotid artery of a goat, injected 35 months after ligation. After *Porta*.

long axis and various transverse axes of the limb, reaches other arteries, through which it soon again streams into the peripheral end of the ligated vessel. An arterial collateral circulation is established to the side of the ligated and thrombosed portion of the arterial trunk. Without this, the part of the body lying below this point would not receive sufficient blood and would die; it would dry up or putrefy. Fortunately, arterial anastomoses are so free that, even after ligation of a large artery, like the axillary or femoral, such a case is not apt to occur; in diseased arteries, however, which do not distend sufficiently, mortification of the affected extremity may occur. The modes in which these new vascular connections form vary greatly. Years ago, *Porta* made very profound researches on this point, and from his numerous experiments stated the following, as the types of collateral circulation:

1. *Direct* collateral circulation is established; i. e., there are strongly-developed vessels, which pass from the central end of the artery directly to the peripheral end.

FIG. 33.



Femoral artery of a large dog, injected 3 months after ligation. After *Porta*.

These uniting vessels are chiefly the dilated vasa vasorum, and the vessels of the thrombus; it might happen that one of these uniting vessels should dilate so much as to acquire the appearance of being simply the trunk regenerated.

2. There is an *indirect* collateral circulation; i. e., the connecting branches of the next lateral arteries are greatly dilated, as in the following case, Fig. 33.

The most striking examples of both varieties of collateral circulation have here been chosen; but when you examine the numerous sketches of *Porta*, and yourselves repeat these experiments, you will find that in most cases direct and indirect collateral circulation are combined, so the only value of the classification is to group the different forms in some way.

It is an excellent anatomical exercise, to represent for yourselves how, after ligation of the different arteries of one or both extremities, or of the trunk, the blood will reach the parts beyond the point of ligation ; in this you would be well assisted by the plates of arterial anastomosis in *Krause's* text-book of anatomy. In the surgery of old *Conrad Martin Langenbeck*, these conditions are carefully described in the chapter on aneurisms. The reversal of the blood-current, which not unfrequently takes place in these collateral circulations, occurs with wonderful rapidity, when the anastomoses are free ; if, for instance, we ligate the common carotid in a man, and then divide the artery beyond the ligature, the blood escapes with great force from the peripheral end, that is, backward as from a vein. In all such cases, where the artery to be ligated has free anastomoses, if a piece is to be cut out of the artery, we should first ligate both central and peripheral ends, to be insured against hæmorrhage ; this is an important practical rule, which is often neglected.

The recent investigations on the formation of vessels by *Arnold*, already spoken of, as well as those on tubercle, of which we shall hereafter speak, have given new material for the view that the substance of the wall of the vessel as well as its endothelium takes an active part in the new formation ; and in a recently published work of *Riedel's* it is stated as very probable that the greater part of the young tissue formed in a thrombus proceeds from the endothelium. Thus the views on this subject have been unsettled for years.

# CHAPTER II.

## *SOME PECULIARITIES OF PUNCTURED WOUNDS.*

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### LECTURE X.

**As a Rule, Punctured Wounds heal quickly by First Intention.—Needle Punctures; Needles remaining in the Body, their Extraction.—Punctured Wounds of the Nerves. —Punctured Wounds of the Arteries: Aneurysma Traumaticum, Varicosum, Varix Aneurysmaticus.—Punctured Wounds of the Veins, Venesection.**

MOST punctured wounds are simple wounds, and usually heal by first intention; many of them are at the same time incised wounds, when the puncturing instrument has a certain breadth; some have the characteristics of contused wounds, when the puncturing instrument was blunt; in this case there is generally more or less suppuration. We make many punctured wounds with our surgical instruments, as with *acupuncture needles*—fine, long needles, that we occasionally employ to examine whether and how deep below a tumor or ulcer the bone is destroyed, etc.; with *acupressure needles*, which we use for arresting hæmorrhage; with the *trocar*, a dagger with a three-sided point, furnished with a closely-fitting canula, an instrument for drawing off fluid from cavities.

Dirk, sword, knife, and bayonet punctures are often simultaneously incised and contused wounds. If these punctured wounds be not accompanied by injury of large arteries, veins, or bones, and do not enter any of the cavities of the body, they often heal rapidly and without treatment.

The most frequent punctured wounds are those made with *needles*, especially in women, and how rarely a doctor is called for them! Such an injury is only complicated by a needle, or a part of one, entering the soft parts so deeply that it cannot readily be extracted. This occasionally happens in different parts of the body, as from a person sitting or falling on a needle, or some such accident. If a needle has entered deep under the skin, the symptoms are usually so slight that the patients rarely have any decided sensation of it; indeed, they often cannot say whether the needle has really entered, and where it is. And in the soft parts this body usually induces no external symptoms, but may be carried in the body for months, years, or even a lifetime, without trouble, if it do not enter a nerve. The needle rarely remains stationary at the point where it entered, but wanders about; it is shoved along to other parts of the body by contraction of the muscles, and thus may come to light a long distance from the point of entrance. Cases have been observed where hysterical women, from the peculiar vanity of attracting the attention of physicians, have inserted numerous needles in different parts of the body; these needles appeared now here, now there. Even when needles have been swallowed, they may without danger pass through the walls of the stomach and intestines, and come to the surface at any part of the abdominal wall. *B. von Langenbeck* found a pin in the centre of a vesical calculus; on more careful inquiry, it was found that, when a child, the patient had swallowed a pin. The pin may have passed through the intestine into the bladder; here triple phosphates were deposited around it in layers, and this was possibly the origin of the calculus. *Dittle* had a similar experience.

When the needle has remained for a time in the soft parts without exciting pain, or when needles, passing through the body from within outward, come to the surface close under the skin, they usually excite a little suppuration; the piercing feeling becomes more decided; we make an incision at the painful spot, let out a little thin pus, and in the pus-cavity find the needle, which may be readily removed with forceps. It is difficult to explain why this body, which for months has moved about in the body, should at length excite suppuration when it arrives under the skin; you must here satisfy yourselves with a simple knowledge of the facts. The following interesting case may render the course of these injuries more clear to you: In Zürich a perfectly idiotic female deaf mute, thirty years old, was brought to the clinic with the diagnosis: typhus. No history of the case could be obtained from the patient or those about her, who were also lacking in intelligence. The patient, who often remained in bed for days, had complained for a short time of pain in the ileo-cæcal region, and had moderate fever. Examination showed a swelling at this point, which increased the following days, and was very painful on pressure; the skin reddened, fluctuation became evident. It was clearly not a case of typhus, but you may imagine what different diagnoses there were as to the seat of the suppuration, for there was undoubtedly an abscess; it might be inflammation of the ovary, perforation of the vermiform process, an abscess in the abdominal walls, etc., etc.; still.

something could be said against all these hypotheses. After a few days the reddened skin became very thin, the abscess pointed about the height of the anterior superior spinous process of the ilium, a few fingers' breadths above Poupart's ligament, and I made an incision through the skin; there was evacuated a gassy, brownish, sanious pus, with a strong fecal odor. As I examined the abscess-cavity with my finger, I felt a hard, rod-like, firm body in the depth of the abscess, and projecting slightly into it. I began to extract it, and pulled and pulled till I brought out a knitting-needle almost a foot long, which was somewhat rusty and pointed down toward the pelvis. The abscess-cavity was clothed with flabby granulations. When I tried to examine the opening that the needle must have left behind, I could no longer find it; it had closed again, and was covered by the granulations. The abscess took a long time to heal; it at last did so without further accident, so that in four weeks the patient was dismissed. As I showed the unfortunate cretin the extracted needle, she laughed in her idiotic way; that was all we could make out of her; perhaps this may have indicated some slight recollection of the needle. It is most probable that the patient had inserted the needle into the vagina or rectum—procedures in which even women not idiotic find some incredible pleasure, as you may see in *Dieffenbach's* operative surgery in the chapter on extraction of foreign bodies. It is not impossible that in this case the needle passed by the side of the vaginal portion of the uterus through the cæcum, for, from the gas-containing pus of the abscess, we may decide that there was at least a temporary communication with the intestine. It is true this cannot be regarded as absolutely certain, for pus in the vicinity of the intestines by the development of stinking gases may putrefy, even when no communication with the interior of the intestines exists or has existed.

The extraction of recently-entered needles may be very difficult, especially as the patients are not unfrequently very undecided in their information about the location of the body, and occasionally from shame will not acknowledge how the needles (in the bladder, for instance) obtained entrance. We should, with the left hand, fix the spot where we shall most probably find the foreign body, carefully endeavoring to press the skin together in folds; we must at the same time be careful that the needle does not again change its position while we are making the incision. Sometimes we feel the body more or less distinctly, and can cause pain by pressing on it; these attempts must decide the point of our incision. After dividing the skin, we attempt to seize the needle with a pair of good dissecting forceps; very tense bands of fascia may readily deceive us, especially about the fingers, for with forceps our sense of feeling is always uncertain.

If we cannot find the needle, we may move the parts some; the needle is then sometimes moved into a position where it may be seized more readily. The extraction of foreign bodies requires a certain amount of practice and manual dexterity, which we acquire only with time and practice; here natural knack is of great service.

Punctured wounds, made with instruments not very sharp, are occasionally interrupted in their process of healing. Externally they heal by first intention, but after a few days there are suppuration and inflammation in the deeper parts; the wound either opens, and the whole tract of the wound suppurates, or the pus breaks through at some other point. This occurs particularly in cases where a foreign body, as the point of a knife, remains behind, or where the wound was made with a blunt instrument. In examining the wound, you should always bear in mind the possibility of a foreign body remaining behind, and, if possible, see the instrument with which the injury was done, and find exactly in what direction the instrument passed, so that you may know about what parts are injured. However, even in unfavorable cases there are occasionally very little inflammation and suppuration. A short time since a man came to the clinic who, a few days previously, had fallen a moderate height from a tree, lighting on his left arm, while engaged clipping the small branches. On the dorsal surface, a few inches below the elbow, the arm was swollen; on the volar surface, just above the wrist, there was a slight excoriation; the arm could be extended and flexed without pain; only pronation and supination were impaired and painful. There was no solution of continuity of the bones of the forearm; the bones were certainly not broken through. At the swollen spot on the dorsal side, an inch below the elbow, immediately under the skin, we could, however, feel a firm body, which could be pressed back somewhat, but it at once returned to its old position. It felt just as if a piece of bone had been broken off lengthwise, and lay close under the skin. Incomprehensible as it must seem for such a detachment of bone to occur by simply falling on the arm, without fracture of the radius or ulna, I nevertheless had the patient anæsthetized, and again made the attempt to press into position the suspected fragment; but it did not succeed. As it lay so close under the skin that it would necessarily have perforated ere long, I made a small incision through the skin to extract it. To our great astonishment, I drew out, not a fragment of bone, but a small branch, five inches long, which was quite firmly held by the two bones of the forearm. It was incomprehensible how this twig could have entered the forearm; but, on more careful examination at the above-mentioned excoriated spot on the volar surface, we found a linear, slit-like wound, which had already closed, through which the body had apparently passed so quickly that the patient had not noticed its entrance. After its extraction the very moderate swelling entirely subsided; the small wound discharged but little pus, and was entirely closed in eight days.

These favorable conditions of punctured wounds have given rise to the so-called *subcutaneous* operations, which were introduced into surgery more particularly by *Stromeyer* and *Dieffenbach*, and consist in passing a pointed, narrow knife under the skin, and dividing tendons, muscles, or nerves, for various purposes of treatment, without making any wound in the skin other than the small punctured wound through which the tenotome is introduced. Under these circumstances the wound almost always quickly closes by first intention, while in open wounds of tendons there is almost always suppuration, often extensive death of the tendon. Of this we shall speak further in the chapter on deformities (Chapter XVIII.).

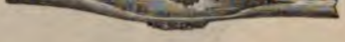
If the puncture has entered one of the cavities of the body, and caused injury there, the prognosis will always be doubtful; there is more or less danger, according to the physiological importance and vulnerability (the greater or less susceptibility to dangerous inflammation) of the organ implicated. Such a punctured wound is not so dangerous as a gunshot wound. We shall not at present pursue this subject further, but must now say something about punctured wounds of the nerves and arteries of the extremities.

Punctured wounds of nerves naturally induce, according to their extent, paralysis of variable amount; otherwise they have the same effect as incised wounds of the nerves. Regeneration occurs the more readily when the whole breadth of the nerve has not been punctured. The case is different when a foreign body, as the point of a needle or a bit of glass, is left in the nerve-trunk; they may heal in here as in other tissues. The cicatrix in the nerve which contains this body may remain excessively painful at every touch; there may also be neuralgia or nervous pains extending excentrically. Moreover, the severest nervous diseases, acute or chronic, may be induced by these foreign bodies. *Epileptiform spasms*, with an *aura*, a pain in the cicatrix preceding the spasm, have been observed after such injuries; some surgeons also assert that *traumatic tetanus* may also be induced by this nervous irritation. This appears to me very doubtful, but of this hereafter. The first of these diseases, the so-called reflex epilepsy, may usually be cured by the extraction of the foreign body.

Punctured wounds of arterial trunks or their large branches may induce various results. A very small puncture usually closes by the elasticity and contractility of the coats; indeed, there is not always a hæmorrhage, any more than there is always escape of fæces from a small puncture of the intestine. If the wound be slit-shaped, the bleeding may also be insignificant if the opening gapes but little; but in other cases severe arterial hæmorrhage is the immediate result. If compression be at once made, and a bandage accurately applied, we shall usually succeed not only in arresting the hæmorrhage, but also in closing the puncture in the artery, just as we should one in the soft parts. If the bleeding be not arrested, as already stated, we should at once ligate the artery, after enlarging the wound up and downward, or at a higher point in the continuity.

The closure of the arterial wound takes place as follows: A blood-clot forms in the more or less gaping wound of the arterial wall; this clot projects slightly into the calibre of the vessel; but externally it is usually somewhat larger, and looks like a mushroom. As described in intra-vascular thrombus, this clot is transformed to connective tissue; and thus there is permanent organic closure, without change of the calibre of the artery. This normal course may be complicated by layers of new fibrine from the circulating blood, depositing on the part of the plug projecting into the calibre of the vessel, and thus closing it by a clot, forming a complete arterial thrombosis; but this is rare. Should it happen, we would have the same result as after a thrombosis following ligation—development of collateral circulation, and eventual obliteration of the vessel by organization of the thrombus.

FIG. 34.

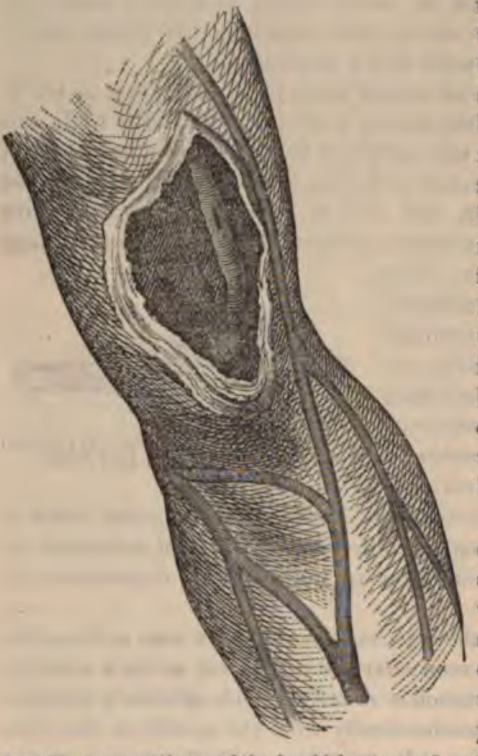


Artery wounded on the side, with clot, four days after the injury; after *Porta.*

Punctured wounds of the arteries do not always take so favorable a course. In many cases, soon after the injury, we notice a tumor at the seat of the young cutaneous cicatrix, which gradually enlarges and perceptibly pulsates isochronically with the systole of the heart and with the arterial pulse. If we place a stethoscope over the tumor, we may hear a distinct buzzing and friction sound. If we compress the chief artery of the extremity above the tumor, the pulsation and murmur cease and the tumor diminishes somewhat. We call such a tumor an *aneurism* (from ἀνευρύνειν, to dilate), and this particular form, arising from wound of an artery, we call *aneurisma spurium* or *traumaticum*, in contradistinction to the *aneurisma verum*, arising spontaneously from other diseases of the artery.

Whence comes this tumor, and what is it? Its origin is as follows: The external wound is closed by pressure, the blood can no longer flow out of it; but it forms a way through the opening, which is not yet firmly closed by the clot, into the soft parts, and winds about among them as long as the pressure of the blood is stronger than the resistance of the tissues; a cavity filled with blood is formed in immediate communication with the calibre of the artery, part of the blood soon coagulates, and there is slight inflammation of the tissue about it; a plastic infiltration, which leads to connective tissue new formation, and this thickened tissue forms a sac, into and from whose cavity the blood flows, while the periphery of the cavity is filled with layers of clotted blood. The buzzing and friction that we perceive in the tumor arise partly from the blood flowing out through the narrow opening in the artery, partly by its friction against the coagulum, and lastly by the regurgitation of the blood into the artery.

FIG. 35.



Aneurisma traumaticum of the brachial artery; after *Froriep*, "Surgical Copperplates." Bd. IV., Plate 483.

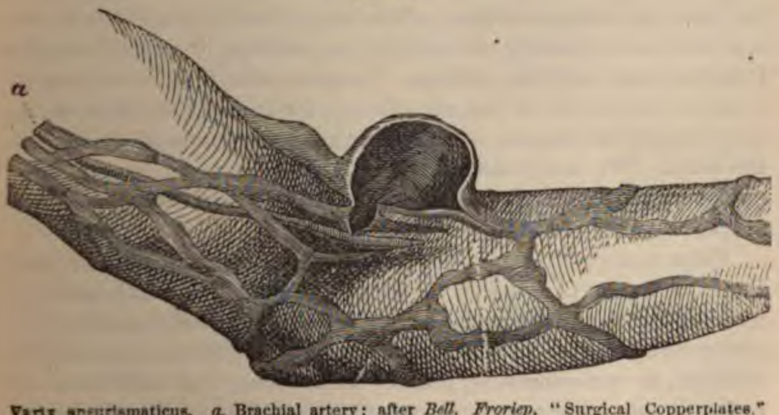
Such a traumatic aneurism may also occur in another, more secondary way; the arterial wound at first heals, and subsequently, after removal of the pressure bandage, the young cicatrix gives way, and then for the first time the blood escapes.

Traumatic aneurisms are not always caused by punctured wounds of arteries, but rupture of their coats by great tension and contusions, without any external wound, may result in their development. Thus, in his surgical lectures, *A. Cooper* tells of a gentleman who leaped a ditch while out shooting, and at the time felt a pain in the hollow of his knee, which prevented his walking. An aneurism of the popliteal artery soon developed in the bend of the knee, that finally had to be operated on. The artery was partly ruptured by the leap. Rupture of the tunica intima and muscularis is sufficient to permit the formation of an aneurism. Should the tunica adventitia remain uninjured, the blood may detach it from the tunica media; this forms a variety of aneurism called *aneurisma dissecans* (dissecting aneurism). Cases of punctured wounds with subsequent aneurisms occur particularly in military practice, but not unfrequently also in civil practice. I saw a boy with an aneurism, as large as a hen's-egg, of the femoral artery, about the middle of the thigh, that had been caused by puncture with a pen-knife, on which the boy fell. A short time since I operated on an aneurism of the radial artery, that had developed in a shoemaker after an accidental puncture with an awl.

*An aneurism is a tumor communicating directly or indirectly with the calibre of an artery.* This is the common definition. The communication is immediate in the case just described of a simple traumatic aneurism. Still, the anatomical conditions of this tumor may be more complicated.

For instance, in a venesection at the bend of the elbow, that is, from intentionally puncturing a vein for the purpose of abstracting blood, besides the vein, the brachial artery may be wounded; this is one of the most frequent causes of traumatic aneurism, or at least was so formerly, when bleeding was more common. In such a case, besides the dark, venous blood, we may readily perceive the bright, arterial blood; the whole arm is at once bound up and the artery compressed, and in some cases the openings in both vessels heal at once without further consequences. But occasionally it happens that this accident is followed by an aneurism; this may have the simple form above described; but the openings in the two vessels may so grow together that part of the arterial blood will flow directly into the vein as into an arterial branch, and must then meet the stream of venous blood. This

FIG. 36.

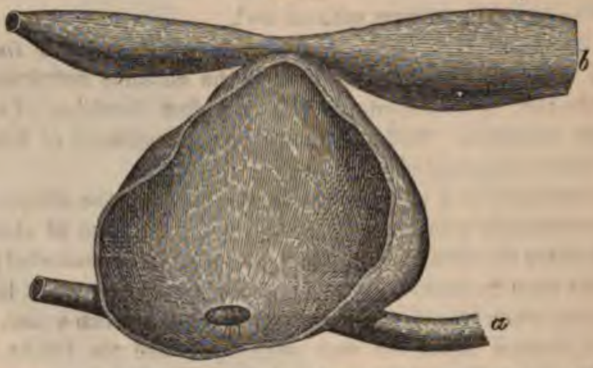


Varix aneurismaticus. *a*, Brachial artery; after *Bell. Froriep*, "Surgical Copperplates." Bd. III., Taf. 263.

causes obstruction of the venous current and consequent sacculations, dilatations of the calibre of the vein, which we generally term *varices;* in this particular case the varix is called *varix aneurismaticus*, because it communicates with an artery like an aneurism.

Another case may arise: an aneurism forms between the artery and vein, both of which communicate with the aneurismal sac.

FIG. 37.



Aneurisma varicosum. *a*, Brachial artery; *b*, median vein. The aneurismal sac is cut open; after *Dorsey*. *Froriep*, "Surgical Copperplates." Bd. III., Taf. 263.

We call this *aneurisma varicosum.* There may also be some varieties in the relation of the aneurismal sac, vein, and artery, to each other, which, however, are only important as being curious, and change neither the symptoms nor treatment, and fortunately have no particular names. In all these cases where arterial blood flows directly or indirectly through an aneurismal sac into the veins, there is distention of the veins and a thrill in them, which may be both felt and heard, and may even be occasionally perceived in the arteries; it probably results from the meeting of the currents. However, this thrill in the vessels is not characteristic of the above state, for it may sometimes be induced simply by pressure on the veins, and occurs in some diseases of the heart. We also occasionally see a weak pulsation in veins distended by the above causes, which would even earlier give a correct diagnosis.

Quite recently I saw a number of aneurisms resulting from gunshot-wounds; in three cases affecting the femoral and external iliac arteries, the above-mentioned thrill was very prominent, rendering it pretty certain that there was a communication between the artery and vein, as was proved by autopsy in one case; but there were no varices in any of these cases; hence their development is not a necessary result of communication between arteries and veins, or else they may in some cases not develop for some years.

Aneurisms of the arteries, in whatever form they come, if they only remained small, would cause no great inconvenience. But in most cases the aneurismal sacs grow larger and larger; functional disturbances occur in the affected extremity, and finally the aneurism may rupture, and a profuse hæmorrhage terminate life. In most cases the treatment must consist in ligating the aneurismal artery; but of this hereafter. I have considered it practical to explain to you here the development of traumatic aneurisms, as in practice they are mostly due to punctured wounds; while in other text-books you will find them systematically treated of among diseases of the arteries. We shall speak, in a separate chapter, of spontaneous aneurisms and their treatment.

*Punctured wounds of veins* heal just like those of arteries, so that I need add nothing here to what was said above; we need only remark here that extensive coagulations form more readily in veins than in arteries; *traumatic venous thrombosis* after venesection, for instance, is far more frequent than traumatic arterial thrombosis after punctured wounds of arteries, and, what is far worse, the former variety of thrombosis has much more serious results than the latter; on this point you will perhaps hereafter hear more than will be agreeable to you.

We have frequently mentioned *venesection*, which is a very frequent small surgical operation. We shall here briefly review its performance, although you comprehend such things quicker and better by once seeing them than I could represent them to you. Should I attempt to tell you under what circumstances venesection should be performed, I should have to enter deeply into the whole subject of medicine; quite a large book might be written on the indications and contraindications, the admissibility, the benefits and injuries of venesection; hence I prefer to say nothing on these points as on so many others which you will pick up in a few minutes at your daily visits to the clinics, and for whose theoretical exposition without special cases we should require hours. In regard to the history, we will only mention that, while formerly venesection was performed on any of the subcutaneous veins, now it is only done in the veins of the bend of the elbow. If you wish to bleed a patient, you first apply a pressure-bandage to the arm, to cause obstruction of the peripheral veins; for this purpose we employ a properly-applied handkerchief or the old-fashioned scarlet bleeding-ribbon, a firm bandage two or three finger-breadths wide with a buckle; when this is firmly applied the veins of the forearm swell up and the vena cephalica and basilica with their corresponding median veins appear in the bend of the elbow. You choose, for opening, the vein which is most prominent. The arm of the patient is flexed at an obtuse angle; with the left thumb you fix the vein, with the lancet or a very pointed straight scalpel in the right hand you puncture the vein and slit it up longitudinally two or three lines. The blood escapes in a stream; you allow sufficient to flow, cover the puncture with your thumb, remove the bandage from the arm above, and the bleeding will cease spontaneously; the wound should be covered with a small compress and a bandage; the arm should be kept quiet three or four days, then the wound will be healed. Easy as this operation is in most cases, it still requires practice. Puncture with the lancet or scalpel is to be preferred to the spring-lancet; the latter was formerly very popular, but is now very justly going out of fashion; the spring-lancet is a so-called fleam, which is driven into the vein with a spring; we allow the instrument to operate, instead of doing it ourselves more certainly with the hand.

Various obstacles may interfere with venesection. In very fat persons it is often difficult to see or feel the veins through the skin; then besides compression we employ another means, that is holding the forearm in warm water, which increases the afflux of blood to this part of the body. Moreover, after opening the vein the fat may impede the escape of the blood by fat-lobules lying in the opening; these should be quickly snipped off with the scissors. Occasionally the flow of blood is mechanically obstructed by the arm being rotated or bent at a different angle after the puncture has been made, so that the opening in the vein no longer corresponds to that in the skin; this is to be met by changing the position of the arm. There are other causes for the blood not flowing properly; such as the puncture being too small, a frequent fault with beginners; the compression is too weak, this may be improved by tightening the bandage; or, on the contrary, the compression is too great, so that the artery is also compressed, and little or no blood flows from the arm, this may be obviated by loosening the venesection bandage. Aids for increasing the flow of blood are: dipping the hand in warm water, and having the patient rhythmically open and close the hand, so that the blood may be forced out by the muscular contractions. We shall speak further on this point, as opportunity offers, in the clinic.

# CHAPTER III.

## *CONTUSIONS OF THE SOFT PARTS WITHOUT WOUNDS.*

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### LECTURE XI.

Causes of Contusions.—Nervous Concussion.—Subcutaneous Rupture of Vessels.—Rupture of Arteries.—Suggillations.—Ecchymoses.—Reabsorption.—Termination in Fibrous Tumors, in Cysts, in Suppuration, and Putrefaction.—Treatment.

By the action of a blunt object on the soft parts, the skin will sometimes be injured, sometimes it will not; hence we distinguish contusions with or without wounds. We shall first consider the latter.

These contusions are partly caused by the falling or striking of heavy objects on the body, partly by the body falling or striking against a hard, firm object. The immediate result of such a contusion is a crushing of the soft parts, which may be of any grade; often we perceive scarcely any change, in other cases the parts are ground to a pulp.

Whether the skin suffers solution of continuity by this application of force depends on various circumstances, especially on the form of the contusing body and the force of the blow, also on the nature of the parts under the skin; for instance, the same force would cause contusion without a wound in a muscular thigh, that applied to the spine of the tibia would cause a wound, for in the latter case the sharp edge of bone would cut the skin from within outward. The elasticity and thickness of the skin also come into consideration; these not only vary in different persons, but may differ in different parts of the body of the same individual.

In contusion without wound we cannot immediately recognize the amount of destruction, but only indirectly from the state of the nerves and vessels, and also from the subsequent course.

In contusion the first symptom in the *nerves* is pain, just as it is in wounds, but pain of a duller, more undefined character, although it may be very severe. In many cases, especially when he has struck against a hard body, the patient has a peculiar vibrating, threatening feeling in the injured part; this feeling, which extends some distance beyond the seat of injury, is caused by the *concussion* of the nerves. For instance, if we strike the hand or finger quite hard, only a small part is actually contused, but not unfrequently there is concussion of the nerves of the whole hand, with great trembling, dull pain, on account of which the fingers cannot be moved, and there is almost complete loss of feeling for the moment. This condition passes off quickly, usually in a few seconds, and then a burning pain is felt in the contused part. The only explanation we have of this temporary symptom is that the nerve-substance of the axis cylinder suffers molecular displacement from the blow, which spontaneously passes off again. These symptoms of concussion (the commotion) do not by any means accompany all contusions; they fail especially in cases where a heavy body comes against a limb at rest, but they are not unfrequently of great significance in contusions of the head; here *commotio* cerebri is not unfrequently united with *contusio* cerebri, or the former appears alone, for instance, in a fall on the feet or buttocks, whence the concussion is propagated to the brain and may induce very severe accidents or even death, without any preceptible anatomical changes. Concussion is essentially a change in the nervous system, hence we speak chiefly of cerebral or spinal concussion. But the peripheral nerves also may be concussed with the above symptoms; but since in such cases the more localized contusion is especially prominent, this nervous state is perhaps too much neglected. Severe concussion of the thorax may induce the most dangerous symptoms simply from concussion of the cardiac and pulmonary nerves, whereby the circulation and respiration are disturbed, although for the most part only temporarily. Nor can a reflex action of the concussed nerve, especially of the sympathetic on the brain, be entirely denied. Doubtless some of you, when wrestling or boxing, have received a blow in the abdomen; what terrible pain! a feeling of faintness almost overcomes you for a time; here we have an action on the brain and on the heart; one holds his breath and gathers his strength, to prevent sinking to the earth. Concussion of the ulnar nerve often occurs, when we strike the elbow hard; most of you probably know the heavy, dull pain, extending even to the little finger. Compression of sensitive nerves is said to cause contraction of the cerebral vessels, as is shown by recent experiments on rabbits; possibly this explains the faintness from severe pain.

All these are symptoms of concussion in the peripheral nerves. Now, as we do not know what specially takes place in the nerves, we cannot judge whether these changes have any effect, and, if so, what, on the subsequent course of the contusion, and of the contused wound; hence we cannot here study the nerves any further. Some unimpeachable observations seem to prove that this concussion of peripheral nerves may induce motor and sensory paralysis, as well as atrophy of the muscles of a limb; but the connection between cause and effect is often difficult to prove.

*Contusions* of the nerves are distinguished from concussions by the fact that in them certain parts of the nerve-trunks, or their whole thickness, is destroyed, to the most varied extent and degree, by the force applied, so that we find them more or less pulpy. Under these circumstances, there must be a paralysis corresponding to the injury, from which we determine the nerve affected, and the extent of the effect. On the whole, such contusions of nerves without wounds are rare, for the chief nerve-trunks lie deep between the muscles, and so are less apt to be injured directly.

It must *a priori* be acknowledged that concussion may affect other organs and tissues than nerves, and induce temporary or permanent disturbances, not only of the functional but of the nutritive processes. Such disturbances may also have an important influence on the course of repair after the injury, and are mentioned by some surgeons as the chief causes of inflammations that are occasionally very violent and develop easily-decomposing exudations and infiltrations. I am far from denying the influence of an energetic concussion on a bone whose medulla and vessels are thereby torn, without its being fractured; under some circumstances the results of such an injury might be more extensive and tedious than those of a fracture from too great bending; but we should not ascribe the frequent severity of the course of contused wounds entirely to this cause.

*Contusions* of the vessels must be much more apparent, since the walls of the smaller vessels, especially of the subcutaneous veins, are destroyed by the contusing force, and blood escapes from them. Hence, *subcutaneous hæmorrhage* is the almost constant consequence of a contusion. It would be much more considerable if in this variety of injury the wound of the vessel had sharp edges, and gaped; but this is not usually the case. Contused wounds of the vessel are rough, uneven, ragged, and these irregularities form obstacles to the escape of the blood; the friction is so great that the pressure of the blood is unable to overcome it; fibrinous clots form on these inequalities, even extending into the calibre of the vessel, causing mechanical closure of the vessel, or thrombus. Contusion of the wall of a vessel, with alteration of its structure, may alone cause coagulation of the blood; for *Brücke* has proved that a living, healthy intima of the vessel is very important for the fluidity of the blood within the vessel. We shall again return to this subject, under contused wounds. The counter-pressure of the soft parts prevents an excessive escape of blood, for the muscles and skin exercise a natural compression; hence, these subcutaneous hæmorrhages, even when from a large vessel of the extremities, are very seldom instantly dangerous to life. Of course, it is different in hæmorrhages into the cavities of the body; here there is little besides movable parts, that can offer no sufficient opposition to the escape of the blood; hence, these hæmorrhages are not infrequently fatal. This may be in two ways: partly from the amount of blood escaping—into the thorax or abdomen, for instance—partly from the pressure of the blood on the parts in the cavity—on the brain, for instance—which are not only partly destroyed by the blood flowing from large vessels, but are compressed in various directions, and their functions thus impaired. Hence, hæmorrhages in the brain cause rapidly-occurring paralyses, and often, also, disturbance of the sensorium. In the brain we call this escape of blood, as well as the symptoms induced by it, apoplexy (from ἀπο and πλησσω, to knock down).

If a large artery of an extremity be contused, the conditions are the same as in a stitched or compressed punctured wound. A traumatic aneurism, a pulsating tumor, forms, as described in the last lecture. But this is rare as compared with the numerous contusions occurring daily, and is so, doubtless, because the larger arteries lie quite deep, and the arterial coats are firm and elastic, so that they tear far less readily than the veins, although a short time since, in the clinic, we saw a subcutaneous rupture of the anterior tibial artery. A strong, muscular man had a fracture of the leg; the skin was uninjured; the tibia was fractured about the middle, the fibula rather higher. The considerable tumor that at once formed at the seat of fracture pulsated visibly and perceptibly to the touch on the anterior surface of the leg. There was very evident buzzing sound in it, which I was able to demonstrate to the class. The foot was dressed with splints and bandages; we avoided the application of an immovable dressing, so that we might watch the further course of the traumatic aneurism that had evidently formed here. We renewed the dressing every three or four days, and could see the tumor gradually becoming smaller and pulsating less strongly, till it finally disappeared, a fortnight after the injury. The aneurism had been cured by the compression from the bandage. Nor was the recovery of the fracture interrupted; eight weeks after the injury, the patient had full use of his limb.

The most frequent subcutaneous hæmorrhages in contusions are from rupture of the subcutaneous veins. These effusions of blood cause visible symptoms which vary, partly from the quantity of the effused blood, partly from the distribution of the blood in the tissue.

The more vascular a part, and the more severely contused, the greater the extravasation. The extravasated blood, if it escapes from the vessels slowly, forms a passage-way between the connective-tissue bundles, especially those of the subcutaneous connective tissue and muscles; this must cause infiltration of the tissue with blood and consequent swelling. These diffuse and subcutaneous hæmorrhages we term *suggillations* or *suffusions*. The more relaxed and yielding, and the easier to press apart the tissue is, the more extensive will be the infiltration of blood, if it flows gradually but continually from the vessels for a time. Hence, as a rule, we find the effusions of blood in the eyelids and scrotum quite extensive, because the subcutaneous connective tissue there is so loose. The thinner the skin, the more readily and quickly we shall recognize the suggillation; the blood has a blue color through the skin, or presses into it and gives it a steel-blue color. Under the conjunctiva bulbi, on the contrary, the blood appears quite red, as this membrane is so thin and transparent. Blood extravasations in the cutis itself appear as red spots (purpura) or striæ (vibices); but in this form they are very rarely due to contusion, they are caused by spontaneous rupture of the vessels; whether because the walls of the vessels are particularly thin in some persons, as in those already mentioned as being of hæmorrhagic diathesis, or because they are especially brittle and tender from some unknown condition of the blood, as in scorbutis, some forms of typhus, morbus maculosus Werlhofii, etc. Contusion of the cutis may usually be recognized by a very dark-blue color, passing into brown; also by striation of the epidermis with so-called *chaps*, or, as they are technically termed, *excoriations*, flaying of the skin.

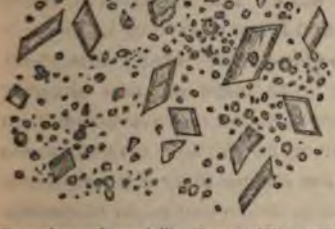
If much blood escape suddenly from the vessels and be effused in the loose cellular tissue, a more or less bounded cavity is formed. This form of effusion of blood is called *ecchymosis*, *ecchymoma*, *hæmatoma*, or blood-tumor. Whether the skin be discolored at the same time, depends on how deep the blood lies under it. In deep effusions of blood, diffuse as well as circumscribed, we often find no discoloration of the skin, especially soon after the injury; we only perceive a tumor whose rapid development immediately after an injury at once shows its nature; this tumor feels soft and tense. The circumscribed effusion of blood offers the very characteristic feeling of *fluctuation*. You may most readily obtain a clear idea of this feeling by filling a bladder with water and then feeling its walls. In surgical practice the recognition of fluctuation is very important, for there are innumerable cases where it is important to determine whether we have to deal with a tumor of firm consistence, or with one containing fluid. You will be shown in the clinic how it is best to make this examination in different cases.

Some of these effusions of blood have received particular names according to the localities where they occur. Thus those coming on the heads of the newly-born, between the various coverings of the skull and in it, are called *cephalhæmatoma* (from κεφαλή, head, and ἁιματόω, to soil with blood), cephalic tumors of the newly-born. The extravasations in the labia majora, from contusions or the spontaneous rupture of distended veins, have received the neat name of *episiohæmatoma* or *episiorrhagia* (from ἐπείσιον, the external genitals). Effusions of blood in the pleura and pericardium have also special designations: *hæmatothorax*, *hæmatopericardium*, etc. On the whole, we attach little importance to these euphonic Latin and Greek names; but you should know them, so as to understand them when reading medical books, and not seek for any thing mysterious behind them; also that you may use them so as to express yourself quicker, and be readily understood.

The subsequent course and symptoms are very characteristic of these subcutaneous effusions of blood. Looking first at the diffuse effusions of blood, immediately after the injury, we are rarely able to decide how extensive the bleeding has been or still is. If you examine the contused part the second or third day after the injury, you notice that the discoloration is more extensive than on the first day; this appears to increase subsequently; that is, it becomes more perceptible. The extent is sometimes astonishing. We once had in the clinic a man with fractured scapula; at first there was only slight discoloration of the skin, although there was a large fluctuating tumor. On the eighth day, the whole back from the neck to the gluteal muscles was of a dark steel-blue, and presented a peculiar, almost comical appearance, the skin looking as if painted. Such widely-spreading extravasations are particularly apt to occur in cases of fractured bones, especially of the arm or leg. But fortunately this partly dark-blue, partly bluish-red color, along with which the skin is not sensitive and scarcely swollen, does not remain so, but further changes take place; first there is further change of color, the blue and red pass into mixed brown, then to green, and finally to a bright lemon yellow. This peculiar play of colors has given rise to the expression of "beating one black and blue," or "giving one a black eye." The last color, the yellow, usually remains a long time, often for months; it finally disappears, and no visible trace of the extravasation remains.

If we ask ourselves whence come these various colorings of the skin, and if we have the opportunity of examining blood extravasations in various stages, we find that it is the coloring matter of the blood which gradually passes through the metamorphoses and shades of color. When the blood has escaped from the vessels and entered the connective tissue, the fibrine coagulates. The serum enters the connective tissue, and thence passes back into the vessels; it is reabsorbed. The coloring matter of the blood leaves the blood-corpuscles, and in a state of solution is distributed through the tissue. The fibrine and blood-corpuscles, for the most part, disintegrate to fine molecules, and in this state are reabsorbed by the vessels; as in the thrombus a few white blood-cells may attain a higher development. The coloring matter of the blood which saturates the tissues passes through various, not thoroughly understood metamorphoses with change of color, till it is finally transformed into a permanent coloring matter, which is no longer soluble in the fluids of the body—*hæmatoidin.* As in the thrombus, this is partly granular, partly crystalline; in a pure state it is orange-colored, and if scanty gives the tissue a yellowish color, if plentiful a deep orange hue.

FIG. 38.



Granular and crystalline hæmatoidin, partly orange, partly ruby-red in color. Magnified 400.

*Reabsorption* of the extravasation almost always takes place in diffuse suggillations, as the blood is very widely distributed through the tissues, and the vessels that have to accomplish the reabsorption have not been affected by the contusion; it is the most desirable and under favorable circumstances the most frequent result after subcutaneous and intermuscular effusions of blood.

The case is different in circumscribed effusions, in *ecchymoses.* Here the first question is as to the extent of the effusion, then about the state of the vessels surrounding it; the more developed the latter, the less they have been injured by the contusion, the more hope there is of early reabsorption; but its occurrence is always less constant in large effusions of this variety. There are various factors which interfere with it; in the first place, there is thickening of the connective tissue around the effusion of blood, as around a foreign body (as in traumatic aneurism also), by which the blood is entirely encapsulated; the fibrine of the effusion is deposited in layers on the inner surface of this sac, the fluid blood remains in the middle. Thus the vessels about the blood-tumor can take up very little fluid, as they are separated from the fluid part of the blood by layers of fibrine, which ar often quite thick. Here we have the same conditions as in large fibrinous exudations in the pleura; there also the fibrous deposits on the walls greatly interfere with reabsorption. This can only take place perfectly when the fibrine disintegrates to molecules, becomes fluid, and thus absorbable; or when it is organized to connective tissue, and supplied with blood and lymph vessels. This is not so very rare in pleuritic deposits. But there is also another fate for such extravasations. The fluid portion of the blood may be completely reabsorbed, and a firm tumor composed of concentric, onion-like layers may remain. This results occasionally from extravasations in the labia majora; a so-called *fibrous tumor* is thus formed; in the cavity of the uterus, also, such fibrous tumors occasionally develop. Some hæmatomata may be partly organized to connective tissue, and gradually take up lime-salts and entirely calcify; a rare termination, but one that occurs in effusions of blood in large goitres. Another mode is the transformation of the blood-tumor to a *cyst;* this is seen in the brain, and in soft tumors. Besides other modes of origin, some cysts in goitres may owe their origin to such effusions. By a cyst or encysted tumor we mean sacs or bags containing more or less fluid. The contents of these cysts, resulting from extravasation of blood, are darker or lighter according to their age; indeed, the blood-red may totally disappear from them, and the contents become quite clear or only slightly clouded by fat molecules. In large circumscribed extravasations you will find numerous and beautifully-formed hematoidin crystals more rarely than in small diffuse ones, for in the former fatty disintegration of the elements of the blood predominates, hence excretion of cholesterine crystals is more common in them. The capsule enclosing these old effusions arises partly from organization of the peripheral parts of the blood-clot, partly from the circumjacent tissue.

*Suppuration* of circumscribed extravasations is far more frequent than the two last described metamorphoses, but is not so common as reabsorption. The inflammation in the vicinity, and the plastic process in the peripheral part of the extravasation, from which, in the two preceding cases, the thickened connective tissue was developed, which encapsulated the blood, assume a more acute character in the case we are about to speak of; a boundary layer is formed here also, but not slowly and gradually as in the preceding cases, but by rapid cell-formation; plastic infiltration of the tissue does not lead to development of connective tissue, but to suppuration; the inflammation after a time attacks the cutis, and it suppurates from within outward, and is finally perforated, and the pus mixed with blood is evacuated; the walls of the cavity come together, cicatrize and grow together, and healing thus takes place. We shall speak more exactly of this mode of healing when treating of abscess; we call any pus-tumor, i. e., circumscribed collection of pus under the skin at any depth, an abscess: hence we term the above process the conversion of an extravasation of blood into an abscess. This process may be very protracted, it may last three or four weeks, but, if not dangerous from its location, it generally runs a favorable course. We recognize the suppuration of an extravasation of blood by the increasing inflammatory redness of the skin, the growth of the tumor, increasing pain, occasionally accompanied by fever, and finally by thinning of the skin at some point, where it is finally perforated.

Lastly, there may be rapid decomposition of the extravasation; fortunately, this is rare. Then the tumor grows hot, tense, and very painful, the fever usually becomes considerable, chills and other severe general symptoms may occur. This termination is the worst, and the only one that requires speedy relief.

Whether there shall be reabsorption, suppuration, or putrefaction of an extravasation, depends not only on the amount of the effused blood, but very much on the grade of the contusion that the tissues have suffered; as long as these may return to their normal state, reabsorption will be probable; if the tissues be broken down and pass into disintegration or decomposition, they will induce suppuration or decomposition of the blood; briefly, the effused blood will have the same fate as the contused tissue.

While the skin is uninjured we cannot judge accurately how much the muscles, tendons, and fasciæ, are injured; occasionally the size of the extravasation may give some aid on this point, but it is a very uncertain measure; it is better to test the amount of functional activity of the affected muscles, but even the results thus given must be carefully accepted; the amount of force that has acted on the part may lead to an approximate estimation of the existing subcutaneous destruction. In contusion of muscles, as in wounds, healing takes place from the crushed muscular elements undergoing molecular disintegration and being absorbed, or by being eliminated with the pus on suppuration of the extravasation, but then there is new formation both of connective tissue and muscle.

The largest extravasations, either diffuse or circumscribed, are usually accompanied by injuries of the bones; but it will be better to consider the injury of the bone in a separate section.

If a portion of the body be so crushed as to be entirely or mostly incapable of living, it becomes cold, bluish red, brownish red, then black; it begins to putrefy; the products of putrefaction enter the neighboring tissues and the blood; the local inflammations, as well as the fever, assume peculiar forms. As this is the same in contusions with or without wounds, we shall speak of it later.

[During the operation for castration, when the cord and its nerves are ligated, even under chloroform, we may notice excessive nervous effect.

In *Golz's* experiment of slapping a frog on the belly, the animal falls at once into collapse, and may even die from paralysis of the heart and consequent cerebral anæmia; venous blood, being no longer drawn in by the heart, collects in the large abdominal veins, which become distended while the lungs and brain receive no more blood. This impairment of the heart's function is referred to reflex action. Irritation of a sensitive nerve may induce not only temporary retardation of the heart's action, but acute cerebral anæmia. This may be shown by the following easy experiment: Fasten a rabbit in proper position and pass a long slender needle through the thorax-wall into the heart, then we may judge of the heart's action by the movements of the needle, this forming a lever with a long handle. The animal bears the operation well, and in a short time the pulsations become regular. If we now approach some irritating substance, as chloroform or ammonia, to the nose of the animal, the pulsations will be temporarily arrested and then will be slower and longer, showing retardation of the heart's action. This experiment may be often repeated; it shows that simple irritation of the trigeminus filaments suffices to induce retardation of the heart's action, which in its turn is followed by cerebral anæmia due to contraction of the cerebral capillaries. What in the rabbit is caused by irritation of the trigeminus is produced in man by irritating any nerve of sensation. Perhaps we may thus explain some cases of faintness, ending in death, which occur after severe pain. Those persons appear particularly prone to faint, that restrain all exhibition of pain; while crying, struggling, etc., by increasing the number and depth of the respirations and hastening the circulation, unintentionally but effectively tend to prevent retardation of the heart's action and cerebral anæmia.

Besides the effect of the contusion on the nerves, which we usually recognize only from the symptoms, there are important anatomical changes. To study these experimentally, contusions have been induced in parts of animals which had been rendered bloodless, otherwise extravasations from the ruptured vessels would prevent microscopical examination. Varied as may be the force causing the injury, the variation shown by the injured tissue is slight, except in the vessels. *Gussenbauer*, in slight contusions, found almost exclusively ruptures in the loose connective tissue accompanying the smaller blood-vessels; where the force was greater, there was also solution of continuity of the intercellular substance of the tissue, while the cellular and fibrous parts of the tissue were more or less separated ; the muscular filaments were in many places bent or torn, with the contractile substance here and there escaping from the ruptures of the sarcolemma. Even where the tissue was entirely mashed, the cellular elements, connective-tissue corpuscles, cells of the walls of the vessels, endothelium, etc., were usually preserved, the destruction affecting chiefly the intercellular substance ; even in organs where it is scanty there is solution of continuity between the cells, but they were themselves well preserved. The idea that in crushed tissue every structure is unrecognizable is wholly incorrect. Hence it is not possible to make several distinct grades with special anatomical characteristics ; in bloodless tissues the changes, corresponding to the injury, only vary in grade, or rather in extent.

The two most important signs of extravasation in contusions are the color of the covering and swelling of the part. We have already spoken of the shade, between red and violet, induced by the vicinity of the blood, and we shall hereafter treat of the subsequent change of shade. Swelling of the contused part depends, first, on the presence of blood extravasation, and also on the escape of lymph from the vessels and tissues. The greater the amount of blood escaped the larger the swelling. But between the swelling and color there is some contrast, for with intense coloration there is rarely much swelling, and the reverse. This is because the considerable swelling is induced by rupture of large vessels which lie deeply, and in such cases the escaped blood only shows dimly through the coverings. The shape of the swelling may give some idea of the blood extravasation ; a regular increase of volume indicates hæmorrhagic infiltration, an irregular one a large effusion in a cavity, a hæmatoma or blood-tumor. In the highest grade of contusion, where the soft parts have been crushed, they often appear as a shapeless mass, enclosed by the skin as in a bag, in which circulation has ceased.

Extravasations, instead of being composed of blood, may in some rare cases be of pure lymph ; these enlarge slowly, lack the discoloration and symptoms of coagulation of the blood ; on puncture there escapes a lemon-colored clear fluid, like that of a hydrocele ; their prognosis and treatment differ essentially from those of blood-tumor. They are reabsorbed with difficulty if at all. They remain fluid for months, because they contain no fibrine, and coagulation of the extravasation is the first step toward reabsorption. But, on the other hand, effusions of lymph do not induce suppuration or decomposition, so their prognosis is not unfavorable.]

The *treatment* of contusions without wound has for its object the conduction of the process to the most favorable termination possible, that is, to reabsorption of the extravasation; when this takes place, the injuries to the other soft parts also progress favorably, as the whole process remains subcutaneous. We here refer solely to those cases where the contusion of the soft parts and the extravasation are the only objects of treatment; where the bone is broken it should be treated first of all, the extravasation of itself would scarcely be an object for special treatment. If called to a contusion that has just occurred, the indication may be to arrest any still continuing hæmorrhage; this is best done by compression, which, where convenient, is to be made by evenly-applied bandages. In North Germany, when a child falls on its head, or knocks its forehead, the mother or nurse at once presses the handle of a spoon on the injured spot to prevent the formation of a blood-bruise. This is a very suitable popular remedy; by the instantaneous compression the further escape of blood is hindered, as is also its collection at one point, because it is compelled by the pressure to distribute itself in the surrounding tissue; an ecchymosis just forming may thus be transformed into a suggillation, so that the blood may more readily be absorbed. You may occasionally attain the same object by a well-applied bandage.

But we rarely see the injury so early, and in the great majority of cases there is also an injury of a bone or joint, and the treatment of the blood-extravasation is a secondary object.

The use of cold, in the shape of bladders or rubber bags filled with ice, or of cold lotions, to which it is an old custom to add vinegar or lead-water, is resorted to as a remedy in recent contusions; it is said to prevent excessive inflammation. But you must not rely too much on these remedies; the means that most aids the reabsorption of blood extravasations is regular compression and rest of the part. Hence it is best to envelop the extremities in moist bandages, and over them apply wet cloths, which are to be renewed every three or four hours. Other remedies, which usually act well in inflammations of the skin, such as mercurial ointment, are of little use here. But I must not forget arnica; this remedy is so honored by some families and physicians that they would consider it unpardonable to neglect prescribing lotions of infusion of arnica, or of water with the addition of tincture of arnica. Faith is mighty; one believes in arnica, another in lead-water, a third in vinegar, as the potent external reabsorbent. In all cases the effect is doubtless simply due to the moisture and the variation of temperature of the skin caused by the compress, whereby the capillaries are kept active, now brought to contraction, now to dilatation, and thus placed in a better state for reabsorption because they are active.

Diffuse blood-extravasations of the skin with moderate contusion of the soft parts are usually absorbed without much treatment. If a circumscribed extravasation does not change considerably in the course of a fortnight, there is nevertheless no indication for further interference. We then paint the swelling once or twice daily with dilute tincture of iodine, compress it with a suitable bandage, and not unfrequently see the swelling gradually subside after several weeks. Should it become hot, and the skin over it grow red and painful, we must expect suppuration; then even the continued application of cold will rarely change the course, though it may alleviate it. Then, in order to hasten the termination of the suppuration, which cannot be avoided, we may apply warm fomentations, either simply of folded muslin wet with warm water or cataplasms; now you quietly await the further course; if the general health be not impaired, but the patient feels pretty well, you calmly await perforation; it will perhaps be weeks before the skin gradually becomes thinner at some point and finally opens, the pus is evacuated, the walls of the large cavity fall together, and in a short time the parts are all healed. At the commencement of this lecture I mentioned a case where, with a fractured scapula, there was an enormous partly diffuse, partly circumscribed extravasation; here there was a strongly-fluctuating tumor, which was not reabsorbed, while the diffuse effusion was rapidly removed; the suppuration did not end in perforation till the fifth week, then one and a half to two quarts of pus were evacuated; a week later this enormous cavity was healed, and the patient left the hospital well. Why we do not here interfere earlier and aid Nature by an incision, we shall consider more closely when we treat of abscesses.

Should the tension of the swelling rapidly increase, however, during the suppuration of the extravasation, and high fever with chills occur, we may suppose that the blood and pus are decomposing, that there is putrefaction of the enclosed fluid. Fortunately, this is rare, and occurs almost exclusively where there is great crushing of the muscles or splintering of the bone. With such symptoms of course the putrid fluid should be quickly evacuated; then you should make a *large* incision through the skin, unless this be forbidden by the anatomical position of the parts; in which case several small incisions should be made at points where the fluid may escape freely and easily. [You now have a cavity filled with granulations and containing decomposed pus and broken-down blood-clots. To disinfect this, carefully empty out the contents and cleanse the sac with a two- to four-per-cent. solution of carbolic acid and dry sponges, then apply a five-per-cent. solution of chloride of zinc, introduce a drainage-tube, and put on a *Lister's* dressing.

Generally, after free opening, cleansing, and disinfecting, the cavity heals rapidly, the walls being already partly covered with granulations which, under the compression, unite and melt together.

We should mention that, in case of extensive mortification of the soft parts after such injuries, amputation may be indicated, but this is rare unless there was at the same time fracture of the bone.

Although the sacs of blood extravasations rarely require opening, it is more frequent with those containing lymph, not on account of suppuration, but to induce healing; as they do not coagulate or absorb, but often increase in size, operation is required in most cases, and it is advisable not merely to puncture the tumor and evacuate the contents, but to open it widely, disinfect, and drain it as above described.]

# CHAPTER IV.

## *CONTUSED AND LACERATED WOUNDS OF THE SOFT PARTS.*

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## LECTURE XII.

Mode of Occurrence of these Wounds; their Appearance.—Slight Hæmorrhage in Contused Wounds.—Early Secondary Hæmorrhages.—Gangrene of the Edges of the Wound.—Influences that effect the Slower or more Rapid Detachment of the Dead Tissue.—Indications for Primary Amputation.—Local Complications in Contused Wounds; Decomposition, Putrefaction, Septic Inflammations.—Contusion of Arteries; Late Secondary Hæmorrhages.

The causes of contused wounds, of which we have to treat to-day, are the same as those of simple contusions, only in the first cases the force is usually greater than in the latter, or the body by which they are induced is of such a form as to divide the skin and soft parts easily, or else parts of the body have been injured where the skin is particularly thin, or lies over parts unusually firm.

The kick of a horse, blow from a stick, bite of an animal or a man, being run over, wounding with blunt knives, saws, etc., are frequent causes of contused wounds. Nothing, however, causes more contused wounds than rapidly-moving wheels and rollers of machinery, cutting-machines, circular-saws, spinning-jennies, and the various machines with cog-wheels and hooks. All of these instruments, the product of advancing industry, do much injury among the operatives. Men and women, adults and children, with crushed fingers, mashed hands, rugged, lacerated wounds of the forearm and arm, are now among the constant patients in the surgical wards of hospitals in every large city. Innumerable persons are thus maimed of fingers, hands, or arms, and many of these patients die as a result of their injuries. If to these you add (what recently is becoming rarer, it is true) railroad injuries, those caused by blasting, building tunnels, etc., you may imagine, not only how much sweat, but how much blood, clings to the many evidences of modern culture. At the same time it is not to be denied that the chief cause of these accidents is the carelessness, often the foolhardiness, of the workman. Familiarity with the dangerous object renders persons at last careless and rash; some pay for this with their lives.

Gunshot wounds also essentially belong to contused wounds; but, as they have some peculiarities of their own, we shall treat of them in a special chapter. Lacerated wounds, and tearing out of pieces from the limbs, we shall consider at the end of this chapter.

Fractures of bones of the most varied and dangerous varieties accompany contused wounds from all the above causes; but for the present we shall leave these out of consideration, and treat only of the soft parts.

In most cases, the appearance of a wound indicates whether it was due to incision or contusion. You already know the character of incised wounds, and I have alluded to some cases where a contused wound had the appearance of an incised one, and the reverse. Contused wounds, like incised, may be accompanied by loss of substance, or there may be simply solution of continuity. The borders of these wounds are generally uneven, especially the edges of the skin; the muscles occasionally look as if chopped; tags of the soft parts, of various sizes, not unfrequently large flaps, hang in the wound, and may have a bluish-red color, from the blood stagnated or effused in them. Tendons are torn or pulled out, fasciæ are torn, the skin, for some distance around the wound, is not unfrequently detached from the fascia, especially if the contusing force was combined with a tearing and twisting. The grade of this destruction of the soft parts of course varies greatly, and its extent cannot always be accurately determined, as we cannot always see how far the contusion and tearing extend beyond the wound; from the subsequent course of the wound we often satisfy ourselves that the contusion extended much further than the size of the wound indicated; that separation of muscles, divisions of fasciæ, and effusions of blood, extended under the skin, which may have been but little torn. It is unfortunate that the skin-wound gives no means of judging of the extent and depth of the contusion, for it renders it very difficult to correctly estimate such an injury at the first examination; while the appearance of the wound gives the laity no idea of danger, the experienced surgeon soon sees the gravity of the case.

Since the injury, especially when due to machinery, is very rapidly done, the pain is not great; and immediately after the injury the pain from contused wounds is often very slight; the more so, the greater the injury and crushing of the parts. This is readily explained by the nerves in the wound being entirely mashed and destroyed, consequently incapable of conducting; moreover, what I told you in the last lecture about local concussion of nerves, the so-called stupor of the injured part, comes into play.

At first sight it seems rather remarkable that these contused wounds bleed little, if any, even if large veins or arteries be crushed or torn. There are well-observed cases to show that, after complete crushing of the femoral or axillary artery, there was absolutely no primary hæmorrhage. It is true, this is rare; in many cases where there is complete solution of continuity of a large artery by a contusion, although there is no spirting stream, there is constant trickling of blood; this, coming from the femoral artery, would speedily cause death. I have already told you how this arrest of hæmorrhage takes place in small arteries, but will make it clearer to you by an illustration. A railroad hand was run over by a locomotive, so that the wheel passed over his left thigh just below the hip-joint. The unfortunate was at once brought on a litter to the hospital; meantime he had lost much blood, and came in very pale and anæmic, but perfectly conscious. After complete removal of the torn clothing, we found a horrible mangling of the skin and muscles. The bone was crushed to atoms, the muscles were partly mashed to pulp, partly hung in tags from the wound, the skin was torn up as far as the hip-joint. At no point of this horrible wound did an artery spirt, but from the depth considerable blood constantly trickled out, and the general state of the patient clearly showed that he had already lost much blood. It was evident that the only thing to be done here was to amputate at the hip-joint, but in the condition the patient then was, this was not to be thought of; the new loss of blood from this severe operation would undoubtedly have been at once fatal. Hence it was, first of all, necessary to arrest the hæmorrhage, which evidently came from a rupture of the femoral artery. I first tried to find the femoral in the wound, while it was compressed above; but all the muscles were so displaced, all the anatomical relations were so changed, that this was not quickly done, hence I proceeded to ligate the artery below Poupart's ligament. After this was done, most of the bleeding ceased, but not entirely, on account of the free arterial anastomosis; and as no regular dressing could be applied, on account of the existing mangling, I surrounded the limb firmly with a tourniquet, close below where I proposed to exarticulate. Now the bleeding stopped; we gave various remedies to revivify the patient; wine, warm drinks, etc., were administered, so that, toward evening, he had so far recovered that his temperature was again normal, and the radial pulse was again good

I should have preferred postponing the operation till the following day, if, in spite of ligature and tourniquet, with the strengthening of the heart's beat, there had not been some bleeding from the wound, so that I feared the patient might bleed to death during the night. Hence, with the able help of my assistants, I exarticulated the thigh as rapidly as possible. During the operation the absolute loss of blood was not great, but it was too much for the already-debilitated patient. At first all seemed to go well; the spirting vessels were all ligated, the wound cleansed, and the patient placed in bed; soon he suffered from restlessness and dyspnœa, which increased, finally convulsions occurred, and the patient expired two hours after the operation. Examination of the femoral artery of the crushed extremity showed the following: In the upper third of the thigh there was a crushed and torn part, comprising about one-third the calibre of the artery. The tags of the tunica intima, as well as the other coats of vessel, and the connective tissue of the sheath, had rolled up into the calibre of the artery, and the blood could only escape slowly; the surrounding tissue was completely saturated with blood. In this case, no clot had formed in the artery, as the escape of blood was still too free to permit this; but, if you imagine that the contusion had affected the entire circumference of the artery, you may understand how the tags of the coats of the vessel pressing into its calibre from all sides might have rendered the escape of the blood more difficult, or even impossible; then a thrombus would have formed, and stopped the vessel, and gradually have become organized, so as to cause permanent closure, just as after ligation. If no hæmorrhage had followed the partial crushing of the artery in this case, if, for instance, the crushing had occurred without an external wound, possibly a clot would simply have formed at the part roughened by the contusion, a thrombus forming from the wall; in this case there might have been crushing of the artery with preservation of its calibre, a result that is said to have been observed.

If you apply the above-described condition of a large crushed artery to smaller arteries, you will understand how there may here more readily be complete spontaneous plugging of the calibre of the vessels partly by in-rolling of the fragile, torn tunica intima, partly by contraction of the tunica muscularis and by the tags of the adventitia, and that consequently bleeding may fail almost entirely in such contused wounds.

Observation of this led a French surgeon, *Chassaignac*, to invent an instrument for crushing off portions of the body; he terms this operation *écrasement*, the instrument he calls an *écraseur*. It consists of a strong metallic ligature, composed of small links, which is to be applied around the part to be removed, and then drawn slowly into a strong metal frame by means of a ratch arrangement. When the instrument is properly used it causes absolutely no hæmorrhage. Little favor as the instrument at first found among surgeons, from their dislike to contused wounds in operative surgery, there is no doubt of its advantages in suitable cases. Wounds caused by *écrasement* usually heal with very little local or general reaction; coincident inflammations occur less frequently with this class of wounds than with pure incised wounds. Nevertheless *écrasement* will always be limited to a small number of operations.

There is another factor for limiting the hæmorrhages in extensive contusions, that is, the weakening of the heart's action caused by the injury, probably due to reflex action. Persons badly injured, besides suffering from loss of blood and injury of the nerve-centres, are usually for a time in a state of numbness or stupor; the word most commonly used to express this state of depression is "shock." The fright from the injury and all thoughts about it, which follow in rapid succession, unite in producing great psychical depression, which has a paralyzing effect on the heart's action. Still, even in persons not greatly affected psychically by the injury, as old soldiers who have often been wounded, or very phlegmatic persons, a severe injury is not entirely without this effect, so that we must suppose that there are purely physical causes for shock. Contusions of the abdomen have an even more depressing effect on the nerve-centres than do those of the extremities, as I have already told you. In this connection the so-called beating-experiment (Klopfversuch) of *Golz* is very interesting: if we repeatedly strike a frog sharply on the belly with the handle of a scalpel, he becomes as it were paralytic; as a result of paresis of their walls, the abdominal vessels distend greatly and take up almost all the blood, so that all the other vessels and even the heart become bloodless, and the latter only contracts feebly.

When the patient has recovered from this state of psychical and physical depression, the heart begins to act with its former or even greater energy, then hæmorrhages may occur from vessels that had not previously bled. This variety of secondary hæmorrhage occurs after operations, when the effect of the anæsthetic has passed off. Hence the patient should be carefully watched at this time, to guard against such secondary hæmorrhages, especially if, from the locality of the injury, there be reason to suspect that a large artery has been injured.

Now we must again examine somewhat more attentively the local changes in the wound.

Although doubtless the processes that take place in the contused wound, the changes on its surface and final healing, must be essentially the same as in incised wounds, still in the appearances in the two cases there are considerable differences. One very important circumstance is, that in contused wounds the nutrition of the edges of the skin and soft parts is more or less extensively destroyed or impaired, or, to express this more anatomically, the circulation and nerve influence in the borders of contused wounds are more or less lost. This at once prevents the possibility of healing by first intention, as this requires perfect vitality in the surfaces of the wound. Hence contused wounds always heal with suppuration.

This observation causes us to introduce sutures or try firm union by plasters very rarely; you may consider this as a general rule. There are exceptions to this rule, which you will only learn exactly in the clinic, and of which I shall only incidentally remark, that occasionally we fasten large, loose flaps of skin in their original position, not because we expect them to unite by first intention, but that they may not from the first retract too much and atrophy to too great an extent.

Granulation and suppuration are esentially the same as in wounds with loss of substance, except that they are slower, and we might say more uncertain at many places. In incised wounds with loss of substance also a thin superficial layer of tissue is occasionally lost, if it be not very well nourished; but this is insignificant as compared with the extensive loss of tissue-shreds that occurs in contused wounds. Many days, often for weeks, tags of dead (necrosed) skin, fascia, and tendons, hang to the edges of the wounds, while other parts are luxuriantly granulating.

This process of detachment of the dead from the living tissue takes place as follows: A cell infiltration and formation of vessels, leading to development of granulations, start from the borders of the new tissue; granulations form on the border of the healthy tissue, and their surface breaks down into pus. With this change to the fluid state as it were the solution and melting of the tissue, of course the cohesion of the parts must cease, and the dead shreds, which previously were in continuity with the living tissue by their filamentary connection, must now fall.

Hence part of the surface of contused wounds almost always becomes *necrosed* (from νεκρος, dead), *gangrenous* (from ἡ γάγγραινα from γραινω, I consume), which are both expressions for parts in which circulation and innervation have ceased, or which are entirely dead. The part where the detachment takes place is technically called the *line of demarcation* of the gangrene. These technical terms, which refer to every variety of gangrene, no matter how it occurs, you must only notice provisionally here. I will try to render this process of detachment of necrosed tissue by suppuration more distinct by means of a diagram.

In the portion of connective tissue represented, suppose *c*, the border of the wound, be so destroyed by the injury that its circulation is arrested and it is no longer nourished; the blood is coagulated in the vessels as far as the shading extends in the diagram. Now cell-infiltration and inflammatory new formation begin at the outer edge of the living tissue, at the border between *a* and *b* where the vessels terminate in loops; these vascular loops dilate, grow, and multiply; in the tissue the infiltration is constantly increased by wandering cells, as if the edge of the wound were here; granulation tissue is formed; this turns to pus, on the surface, that is, close to the dead tissue, and then of course the necrosed part falls, because its cohesion with the living tissue has ceased. Hence detachment of the necrosed shreds of tissue results from inflammation with suppuration; when the dead portion of tissue has fallen, the subjacent, suppurating layer of granulations comes to light, having been already developed before the detachment of the necrosed part. What you here see in connective tissue is true of the other tissues, bone not excepted.

FIG. 39.

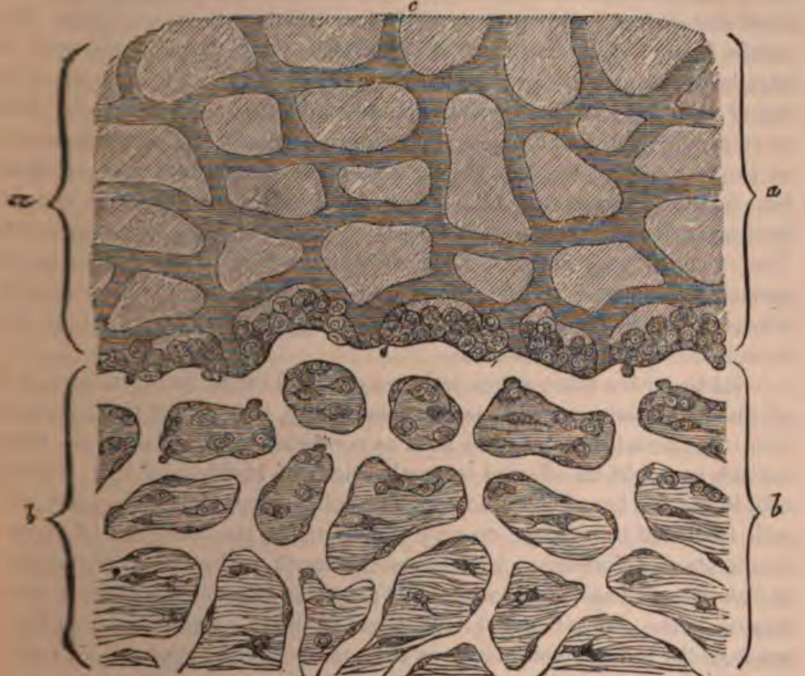

Diagram of the process of detachment of dead connective tissue in contused wounds. Magnified 300 diameters; *a*, crushed necrosed part; *b*, living tissue; *c*, surface of the wound.

In many cases, on the fresh borders of the wound we may see about how much will die, but by no means in all cases, and we can never decide from the first as to the bordering line of the dead tissue.

Completely crushed skin usually has a dark-blue violet appearance and feels cold; in other cases we at first see no change in it, but in a few days it is white, without sensation, later it becomes gray, or, when quite dry, grayish or brownish black. These various colors depend chiefly on the amount of coagulated blood remaining in the vessels or infiltrated in the tissue itself by the partial rupture of the vessels. The healthy skin is bordered by a rose-red line which loses itself in a diffuse redness; this is due to collateral dilatation of the capillaries, and is partly also a symptom of fluxion, of which we have before spoken; it is the reaction redness about the wound, which we have already described; for the living wound-surface only begins where the blood still flows through the capillaries.

In muscles, fasciæ, and tendons, we can decide far less frequently, and often not at all, from the appearance at first, how far they will be detached.

The time required for the dead tissue to be separated and detached from the living varies greatly with the different tissues. This depends first on the vascularity of the tissues; the richer a tissue in capillaries, the softer it is, the more readily cells spread in it, and the richer it is by nature in cells capable of development, so much the more rapidly will the formation of granulations and the detachment of the necrosed parts come about. All these circumstances combine best in the subcutaneous cellular tissue and in the muscles, least so in tendons and fasciæ; the cutis stands in the middle in this respect. The circumstances are the most unfavorable for the bones; consequently the separation of the dead from the living takes place most slowly. Of this more hereafter. Rich supply of nerves seems to have little effect in this process.

But there are many other influences that hinder the detachment of the dead parts, or, what is the same thing, that retard the formation of granulations and pus; such as continued action of cold on the wound, as might be effected by applications of bladders of ice. The cold keeps the vessels contracted. The cell-movements, the escape of cells from the vessels, go on very slowly under the influence of low temperature. Treatment by continued warmth, as by the application of cataplasms, has the opposite effect; by this means we increase the fluxion to the capillaries and cause them to dilate, as you may readily see from the redness you induce on the healthy skin by application of a hot cataplasm; it is known that the high temperature also hastens the cell-activity.

It is entirely impossible to tell beforehand the influence of the general state of the patient on this local process. It is true we may say in general terms that it is energetic in the strong, stout, and young, more moderate and sluggish in weak persons; but on this point we are often deceived.

From what has already been said you may suppose that contused wounds need much longer to heal than more simple incised ones. It will also be evident that there may be circumstances under which amputation of the limb will be necessary, all the soft parts being entirely mashed and torn. There are cases where the soft parts are so torn from the bone that this alone remains; so that on the one hand cicatrization cannot occur, and on the other, if the extremity did heal in months or years, it would be perfectly useless, and hence it would be better to remove it at once. Still, even the simple complete detachment of the skin from the greater part of an extremity may sometimes, though rarely, render amputation necessary, as in the case of a girl who lost the skin from the wrist to the ends of her fingers between the rollers of a spinning machine.

Fortunately such cases are not frequent; in similar injuries of single fingers we mostly leave the detachment to nature; so that no more is lost than is absolutely incapable of living; for we should always remember in maiming of the hand that every line, more or less, is of importance, that especially single fingers, and particularly the thumb, should be preserved whenever possible, for such fingers, if only slightly capable of performing their functions, are more useful than the best-made artificial hand; for the foot and lower extremity there are other considerations, of which we shall hereafter speak when we come to complicated fractures of bones.

Would that this maiming and slow healing, bad as they are, were the only cares we had with our patients having contused wounds! Unfortunately there is a whole series of local and general complications which directly or indirectly endanger life. We shall first speak of the chief local complications; for the more general, the "accidental diseases in wounds," we reserve a future chapter.

Considerable danger may arise from the decomposing tissue on the wound infecting the healthy parts. Putrid matters act as ferments on other organic combinations, especially on fluids containing them; they induce progressive decomposition. We might wonder that such extensive decomposition of the part which is injured, if not killed, should not occur more frequently than it actually does. But in most cases cell-action occurs so quickly on the border of the living tissue that a sort of living wall is formed; this new formation does not readily permit the passage of putrid matter, and the granulation surface, if once formed, is particularly resistant to such influences. In many places it is a popular remedy to cover ulcers with cow-dung and other dirty things; this never causes extensive putrefactions on granulating wounds. But if you apply such substances to fresh wounds, and bind them firmly on so that the tissue may be mechanically impregnated with putrid matter, they will usually become gangrenous to a certain depth, and then an energetic cell-formation opposes the putrefaction. The reason why decomposing matters act so injuriously on fresh wounds, and so slightly on granulating ones, I consider to be, that they are chiefly absorbed by the lymphatic vessels. If you inject a drachm of putrid fluid into the subcutaneous cellular tissue of a dog, the result will be inflammation, fever, and septicæmia. If you make a large granulating surface on a dog, and dress it daily with charpie soaked in putrid fluid, it will have no decided effect. Certain dissolved putrid matters may pass through the walls of the veins and capillaries; but surgical experience teaches that lymphangitis accompanies poisoned wounds much oftener than phlebitis does.

The more the tissue is saturated with fluid, the more it is disposed to decomposition. Hence, the cases where great œdematous swelling occurs after contusions are the most dangerous in this respect; but this œdema comes on very readily as the venous circulation is obstructed, from extensive rupture and crushing of the vessels, which indeed often extend far beyond the borders of the wound.

Imagine a forearm caught under a stone weighing several hundred-weight; there will probably be only a small skin-wound, but extensive crushing of the muscles, tendons, and fasciæ of the forearm, and mashing and rupture of most of the veins; great œdematous swelling will speedily result, as the blood from the arteries is driven with greater energy into the capillaries, and cannot escape by its customary passage through the veins, and hence, under the increased pressure, the serum escapes through the capillary walls into the tissue in greater amount. What a tumult in the circulation and in the whole nutrition! It must soon appear where the blood can still circulate, and where not. In the wound, at first, under the influence of the air, decomposition of the parts incapable of living begins; this advances to the stagnating fluids, and, in unfortunate cases, it constantly progresses; the whole extremity swells terribly as far as the shoulder; the skin becomes bright red, tense, painful, covered with vesicles, from the escape of serum from the cutaneous capillaries under the epidermis. These symptoms usually appear with alarming rapidity the third day after the injury. As a result of this disturbance of circulation, the whole extremity may become gangrenous; in other cases, onlv the fasciæ, tendons, and some shreds of skin die.

There is cell-infiltration of all the connective tissue of the extremity (of the subcutaneous cellular tissue, the perimysium, neurilemma, sheaths of the vessels, periosteum, etc.), which leads to suppuration. Toward the sixth or eighth day the whole extremity may be entirely saturated with pus and putrid fluid. Theoretically, we might imagine such cases curable; that is, we might imagine that, by making suitable openings in the skin, the pus and dead tissue might be evacuated. But this rarely occurs in practice. If the case has undergone the above distention, generally only quick amputation can save the patient, and even this is not always successful. We may term this variety of infiltration sanio-serous. There is a cellular-tissue inflammation, caused by local septic infection; a *septic phlegmon*, whose products again have great tendency to decomposition, but which finally leads to extensive suppuration and necrosis of tissue if the patient lives through the blood-infection which always accompanies it. The earlier such processes limit themselves, the better the prognosis; with the advance of the local symptoms the danger of death of the patient increases.

[We have described the course of a contused wound without treatment; fortunately, under our present method, such results are rare. The progressive putrefaction seems due to decomposition of the dead tissue and fluids, the extravasated blood and lymph. If this be prevented we escape the local effect of the decomposition, and the inflammation is limited to elimination of the mortified tissue, and the general reaction will be inconsiderable. These theoretical conclusions prove correct in practice if the wound can be kept aseptic, that is, putrid decomposition in it prevented. Although good results had been attained by the open treatment, it was properly *Lister's* method that allowed us to follow accurately the undisturbed course of contused wounds. This showed that in them, as in other traumatisms, the injury itself did not induce all the local and general reactions, but that the dangerous symptoms were due to accidental but frequent complications. The first of these is decomposition of the primary secretions from the wound, which are particularly abundant in contused wounds; the crushed soft parts, whose circulation is arrested, furnish excellent material for development of putrefaction. We have already mentioned that decomposition depends on the presence and development of minute germs. These pass into the wound with the air or otherwise, perhaps at the time of the injury, and, as circumstances favor their development by the supply of nourishment, the multiplication of cocci goes on most rapidly, and the decomposition thus induced is favored by the warmth and moisture, just as it would be if we placed dead flesh in a hatching-box supplied with plenty of moist air. Hence it is very important, in treating contused wounds, to prevent infection from without, and to render innocuous any exciters of decomposition already present. This being done, these wounds act like others. It is true, healing by first intention is prevented by the edges of the wound being often in an unviable condition, so that they must be detached. But even to this there are many exceptions; even where the circulation appeared abolished it may be reëstablished, and the edges of the wound will not die, merely a thin layer being detached by molecular disintegration, and union may take place partly by first intention, partly by granulation, but with very little suppuration.

In other cases, if the extravasation be abundant and circulation be arrested in large portions of tissue, healing by first intention is prevented, but by aseptic treatment the extravasated blood may be reabsorbed or become organized to connective tissue; the necrosed parts remain free from smell, are detached slowly without much suppuration; part of the tissue on the border of the dead and living tissue is taken up by the vessels, the remainder of the surface produces healthy granulations, which effect the detachment of the necrosed tissue. In this way even considerable contused wounds may heal with very little inflammatory reaction or suppuration; this may require a long time if the parts are not well supplied with vessels, as fascia, tendons, etc. The dead tissues assume a grayish-brown, tanned look, they dry up from loss of water and are thrown off as small shreds or as molecular *detritus* in the secretion of the wound. Under aseptic treatment this secretion is odorless, at first bloody serum, later more purulent.]

An important thing in the course of contused wounds is the action of the arteries.

An artery may be contused, so as not to be fully divided, and the blood continues to flow through it although part of its wall is incapable of living, and becomes detached on the sixth to the ninth day, or even later. As soon as this occurs, there will be a hæmorrhage in proportion to the size of the artery. These late secondary hæmorrhages, which usually come on suddenly, are exceedingly dangerous, as they attack the patient unexpectedly, sometimes while sleeping, and frequently remain unnoticed until much blood has escaped. Besides the above manner, late arterial secondary hæmorrhage may also result from suppuration of the thrombus, or of the wall of the artery. I observed a case of this kind late in the third week after a severe operation in the immediate vicinity of the femoral artery, in which, however, the artery was not wounded. The bleeding began at night; as the wound looked perfectly well, and the patient had for some time slept the whole night, and for some days had been promised permission to get up the next day, there was no nurse in his private room. He woke in the middle of the night (the twenty-second day after the operation), found himself swimming in blood, and rung at once for the nurse. She instantly called the assistant physician of the ward, who found the patient unconscious. He at once compressed the artery in the wound, and, while I was being called, everything was done to restore the patient. I found him pulseless, unconscious, but breathing, and the heart still beating. While I made ready to ligate the femoral artery, the patient died; he had bled to death. A very sad case! A man otherwise healthy, strong, in the bloom of life, near recovery, must end his life in this miserable way! Rarely has a case so depressed me. Still there was no blame anywhere; as it happened, all the circumstances had been very favorable. The nurse was awake in the next room, the physician was only down one flight of stairs in the same house, and was with the patient in three or four minutes; but the bleeding must have existed before he woke. He was probably awakened by the feeling of wetness in the bed. On autopsy, a small spot of the femoral artery was found suppurated and perforated. Fortunately, it is not always a femoral that bleeds, nor does the bleeding always come so precipitately, or at night; hence, we should not become dissatisfied with our art from such a rare case. Usually such arterial hæmorrhages from suppurating wounds are at first insignificant, and soon cease under styptics or compression; but after a few days the bleeding comes on more actively, and is more difficult to arrest; finally, the hæmorrhages recur more and more quickly, and the patient constantly becomes worse. In all severe arterial hæmorrhage instantaneous compression is the first remedy.

[In contused wounds, secondary hæmorrhages would be actually the rule if the arteries and veins were not closed early by thrombi. This thrombosis is partly due to the contusion, partly to the walls of the vessel not being in condition to preserve the blood fluid, but still not being so much injured as to die; the compression of the vessels by the surrounding tissue may also favor the extension of the thrombosis, which often goes beyond the portion of vessel actually crushed.]

In all arterial secondary hæmorrhages the first remedy to be used is compression on the wound, or, if this is covered with a dressing, on the main artery of the limb; every nurse should know how to do this until an elastic bandage can be applied; and *after* this the surgeon should be sent for. Of course the nurse should not leave the patient for this purpose.

Compression is only a palliative remedy. The bleeding may cease after it ; but, if it be considerable, and you are sure of its origin, I strongly advise you at once to ligate the artery at the point of election, for this is the only certain remedy. You should do this the sooner if the patient be already exhausted ; remember that a second or third such bleeding will surely cause death. Hence, in the operative course, you should particularly practise ligating the arteries, so that you may find them so certainly that you could operate when half asleep. In these particular cases much time is unnecessarily lost in applying styptics, which usually act only palliatively, or not at all. Ligation of arteries is only a trifle for one who knows anatomy thoroughly, and has employed his time well in the operative courses. Anatomy, gentlemen ! Anatomy, and again anatomy ! A human life often hangs on the certainty of your knowledge in this branch.

While treating of secondary hæmorrhages, we shall speak of *parenchymatous hæmorrhages.* The blood rises from the granulations as from a sponge ; we nowhere see a bleeding, spirting vessel. The whole surface bleeds, especially at every change of the dressing. This may be due to various causes : great friability or destructibility of the granulations, that is, their defective organization, may be the fault, and this malorganization of the granulations again may depend on constitutional diseases (hæmorrhagic diathesis, scorbutis, septic or pyæmic infection). Still, local causes about the wound are imaginable, as, if extensive coagulation gradually formed in the surrounding veins, the circulation in the vessels of the granulations would be so affected ; the pressure of blood would so increase that not only the serum might escape from them, but they would rupture. It is true I have hitherto had no opportunity of confirming this by autopsy, but I have seen very few of these parenchymatous hæmorrhages. The latter explanation sounds very plausible ; so far as I know, it originates with *Stromeyer.* He calls such hæmorrhages "hæmostatic." According to the causes, it may be more or less difficult to arrest such hæmorrhages ; in most cases ice, compression, and styptics, will be proper, or, in severe cases, ligation of the arterial trunk, although this occasionally fails. This form of hæmorrhage occurs chiefly in very debilitated persons, who have been exhausted by suppuration and fever, and hence has a bad significance for the general state of the patient.

# LECTURE XIII.

Progressive Suppuration starting from Contused Wounds.—Secondary Inflammations of the Wound: their Causes; Local Infection.—Febrile Reaction in Contused Wounds; Secondary Fever; Suppurative Fever; Chill; their Causes.—Treatment of Contused Wounds: Immersion, Ice-bladders, Irrigation; Criticism of these Methods.—Incisions. — Counter-openings. — Drainage. — Cataplasms. — Open Treatment of Wounds.—*Lister's* Dressing.—Prophylaxis against Secondary Inflammations.—Internal Treatment of those severely Wounded. — Quinine. — Opium. — Lacerated Wounds; Subcutaneous Rupture of Muscles and Tendons; Tearing out of Muscles and Tendons; Tearing out of Pieces of a Limb.

[WE have already stated that by proper antiseptic treatment it is possible, even in severe contused wounds, to reduce inflammation and suppuration to a minimum by preventing decomposition of the secretion of the wound and of the crushed tissues. For this to be possible the case must come under treatment early, but if no rational treatment has been used severe local and general disturbances may occur, even if the injury seems insignificant; in fact, it is these milder cases, where the fingers, hand, etc., have been injured in such a way as not to prevent their use, that may develop the severest symptoms, because the patient uses the part till he has to give it rest.

Let us first consider these dangerous symptoms which, fortunately, are growing rarer from year to year, as antiseptic treatment is becoming more popular. What is the cause of the severe local and general symptoms in the course of contused wounds?]

The granulating surface that develops on a contused wound is generally very irregular, and often has numerous angles and pockets; there is suppuration not only of the surface of the wound, but of the surrounding contused parts under the uninjured skin; hence the neighboring skin often appears undermined by pus. The inflammation and suppuration often unexpectedly extend between the muscles, along the bones, and in the sheaths of the tendons, because these parts were also affected by the injury. The process of inflammation, once excited, creeps along, especially in the sheaths of the tendons and in the cellular tissue; new collections of pus form, superficially or in the depths; the injured part remains swollen and œdematous; on the surface the granulations are smeary, yellow, swollen, and spongy. When we press in the vicinity of the wound, the pus flows slowly from smaller or larger openings, which have formed spontaneously, and this pus which has remained for a time in the depth is not infrequently thin and bad smelling. Should the process continue long, the patient becomes more miserable and weak; he has high and continued fever. A wound, which perhaps at first appeared insignificant, perhaps about the hand, has extended horribly, and induced severe general disturbance. The sheaths of the tendons about the hands and feet are particularly favorable for the extension of deep suppurations, which readily attack the joints, while, on the other hand, articular inflammations of the extremities readily attack the sheaths of the tendons. These states may take a very dangerous turn, and you should be constantly on your guard. From the constant purulent infection, as well as from the daily loss of pus, even the strongest man may emaciate in a few weeks, and die with symptoms of febrile marasmus.

We now know two forms of inflammation which may attack contused wounds: 1. Rapid, progressive, septic inflammation, which begins about the wound during the first three or four days (rarely in less than twenty-four hours, and just as rarely after the fourth day), and which is caused by local infection from parts that decompose in the wound. 2. Progressive purulent inflammation, which is particularly apt to occur in wounds of the hands or feet during the cleansing of the wound from necrosed shreds of tissue, without the pus becoming ichorous, although butyric acid often formed in it.

But, even when the wound has entirely cleaned off and granulated, when the inflammation is bounded, and the wound begins to cicatrize, new inflammation, with severe results, *may* begin. These secondary progressive inflammations of suppurating wounds, occurring even several weeks after the injury, and sometimes coming as unexpectedly as lightning from a clear sky, are of great importance, and are sometimes very dangerous. They are almost always of suppurative nature, and may be fatal from intense, phlogistic, constitutional infection, just as often as the primary progressive suppurations. In some cases, also, they prove dangerous from their location, as in wounds of the head. These cases are so striking and tragical that we must give them special consideration. Suppose you have brought a case of severe crushing of the leg, with fracture, successfully through the first dangers. The patient has no fever; the wound granulates beautifully, and has even begun to cicatrize. Suddenly, in the fourth week, the wound begins to swell; the granulations are croupous or spongy, the pus thin; the whole limb swells. The patient again has high fever, perhaps repeated chills. The symptoms may pass off, and every thing go on in the old track; but it often turns out badly. In a few days the strongest man may become a corpse. Some time since such a case occurred in Zürich, in a fellow-student with a wound of the head; it may serve you as a warning example. The young man received a blow over the left vertex; the bone was injured very superficially; the wound healed quickly by first intention; only a small spot continued to suppurate. As the patient felt quite well, he paid no attention to the little wound, and went about as if perfectly well. Suddenly, in the fourth week, after a walk, he had severe headache and fever. The following day there was about a teaspoonful of pus collected under the cicatrix, which was evacuated by an incision. This did not have the desired beneficial effect on the general condition; the fever remained the same. In the evening delirium began, then sopor. The fourth day the previously vigorous man was dead. It was easy to diagnose that there had been suppurative meningitis. This was proved on autopsy. Although at the spot, as big as a pea, where slight suppuration had been so long kept up, the bone was but slightly discolored by purulent infiltration, still the suppuration on, in, and under the dura mater was greatest at the part exactly corresponding to this point; so that the new inflammation undoubtedly started from the wound. A short time since, here in Vienna, in private practice, I saw a perfectly similar case, also fatal, in a man who several weeks previously had received an apparently insignificant wound, from a piece of a soda-water bottle that burst, at the upper part of the forehead, along the margin of the hairy scalp.

The inflammations occurring under such circumstances, as already remarked, are usually of a diffusely purulent character, but other forms accompany it, or occur spontaneously, such as diphtheritic inflammation of the granulations (*traumatic diphtheria*, *hospital gangrene*), inflammation of the lymphatic trunks (*lymphangitis*), and a specific form of capillary lymphangitis of the skin, *erysipelas* or erysipelatous inflammation; and, lastly, inflammation of the veins (*phlebitis*). Not infrequently all of these processes may be seen mixed together. We shall hereafter study these diseases more accurately, under accidental traumatic diseases. But here we must consider the *causes* of these secondary inflammations, before passing to the treatment of contused wounds; and, in so doing, we must anticipate somewhat. All of these forms of inflammation, and their reflex action on the organism, are so intertwined, that it is impossible to speak of one without mentioning the other.

As causes of secondary inflammations in and around suppurating wounds that have begun to heal, we may mention the following: **1.** Excessive flow of blood to the wound, such as may be induced by too much motion of the part, or by great bodily exertion, as well as by exciting drinks, mental agitation, in short, by any great excitement; in wounds of the head, such congestions are particularly dangerous. Congestion, as caused by too tight bandages, may prove injurious in the same way. 2. Local or general catching cold; about catching cold as a cause of inflammation we know little more than the simple fact that, under certain circumstances, which cannot be accurately defined, a sudden change of temperature induces inflammations, especially in a *locus minoris resistentiæ* of an individual; in a wounded person the wound is always to be considered as a *locus minoris resistentiæ.* The danger of catching cold after injury was certainly over-estimated formerly; I hardly know of any certain examples. 3. *Mechanical irritation of the wound.* This is very important. The pus from the wound is never reabsorbed by the uninjured granulations; but, if they be destroyed by mechanical manipulations, as by improper dressings, much probing, etc., which cause the wound to bleed frequently, new inflammations may be induced. Any foreign bodies in the wound might prove serious in this way, such as pieces of glass, lead, or iron, or sharp splinters of bone; for the first changes which take place in the wound, the vicinity of such foreign bodies is less important, but, when, from muscular movements, and the motion communicated to the tissue from the arteries, the sharp angles of a foreign body keep up constant friction in a part, severe inflammation occurs after a time. 4. *Chemical ferments;* here I mention first soft foreign bodies, such as pieces of clothing, paper wads, which have entered the tissue through gunshot wounds; these substances become impregnated with the secretions from the wound, then the organic material (paper, wool) decomposes, and acts as a caustic and ferment in the wound. I am inclined to believe that necrosed splinters of bone also act rather as chemical than as mechanical irritants; in the Haversian canals, or medullary cavity, they always contain some organic decomposing substance; all such pieces of bone have a putrid smell when extracted; if the surrounding granulations were partly destroyed by the sharp angles of such a fragment of bone, the putrid matter passes from it into the open lymphatic vessels, or possibly even into the blood-vessels, and so induces, not only local, but, at the same time, constitutional infection. Necrosed tags of tendon and fascia at the bottom of suppurating wounds may induce the same results, although this rarely happens. In hospitals, especially, there are some rare cases where we can find none of the above causes; such occurrences naturally induce peculiar alarm, and attempts have been made to explain them by certain injurious influences of the hospital atmosphere, especially such as is filled with the smell of pus. Many circumstances speak against the view that the injurious substances are gaseous; by good ventilation the air of the hospital may be kept pure, but this is no protection against the affection in question; moreover, we cannot excite inflammations by any of the gases developing from pus or putrefying substances, unless, perhaps, by sulphuretted hydrogen, when dissolved in water and injected into the subcutaneous cellular tissue. Putrid fluids and pus from other patients would not intentionally be brought in contact with wounds; we have previously shown that the vicinity of the wound may, under some circumstances, be infected by pus from the wound, and excited to new inflammation. Hence there is little left but the supposition that the injuriously-acting substances are of a molecular, dust-like nature; they may float about in the air of the hospital, but they may also adhere to the bandages, charpie, compresses, etc., with which we dress the wounds, or to the instruments, forceps, probes, sponges, etc., with which we touch the wound. May they not be fungi, or other organic germs, whose nature we do not at present know, like those we know to excite fermentation? This is possible, for in every cubic foot the air holds quantities of such germs, and in the hospital such organic germs of animal or vegetable nature might develop in the secretions from wounds, in the sputum or excrement, and the more so in proportion as the readily-decomposing secretions and excretions are collected in hospitals, or in badly-built water-closets and sewers. On this point we can only hazard conjectures, while we may make experiments with dry putrid substances and dry pus, by powdering them, and then introducing them into the healthy tissue of animals. Such experiments have been made by *O. Weber* and myself, and they have shown that both animal and vegetable putrid, dry substances, as well as dry pus, induce inflammation; if we pulverize these substances, stir them up quickly with water, then inject them into the subcutaneous cellular tissue of animals, they will excite progressive inflammation, just as putrid fluids and fresh pus do. Now, it must at once be acknowledged that in a hospital such injurious dust-like bodies may readily cling to dressings and bedclothes; possibly, also, to instruments. In short, it is possible that the direct injurious influence of hospital air on a wound may be due to fine dust-like particles of putrid or purulent matter coming in contact with it from the dressings or instruments. There can be no doubt that such injurious materials may enter the body in other ways besides through wounds, as through the lungs; indeed, we explain the occurrence of all so-called infectious diseases by the entrance in the organism of substances which have a sort of fermenting influence on the blood; but, whether the morbid materials which excite the infectious diseases chiefly occurring in the wounded be different from those arising from the wound itself, may be a disputed point, so far as we at present know. We shall return to this point when speaking of accidental traumatic diseases. You will suspect me of contradicting myself here, because in yesterday's lecture I said that no molecular body could enter the tissues through an uninjured granulation-surface. I must still claim this as usual; a strong, uninjured granulation-surface is a decided protection against infection through the wound. But, when the infecting material itself is very irritating, so that it destroys the granulating surface by causing decomposition, a passage-way is opened for the poison to enter the tissues. Still more, there are certain substances which are carried into the granulation-tissue, and perhaps even further, by the pus-cells. If you sprinkle a granulating surface on a dog with finely-powdered carmine, some cells take up the small carmine granules and wander with it into the granulation-substance; after a time you find cells with carmine in the granulation-tissue. I consider this an abnormal retrograde movement of the pus-cells, which we generally believe to pass from the granulation-tissue to the surface of the wound; it is true, no one has seen this. Nevertheless, from the above experiment, it is evident that even molecular substances *may* pass from without into the tissue of the edges of the wound, and, if these substances be very decomposable or cauterant, they will excite active inflammation. But all of the millions of molecular organisms in the atmosphere are not taken up by the wound, nor do they each induce inflammation. My belief is that all micrococci do not necessarily have a phlogogenous action, but only those which are formed in certain products of inflammation, such as decomposing pus or fluids of the body, putrid urine, etc., and which have there absorbed the ferment. This is the most frequent cause of micrococcus in hospital; hence its development there is to be combated with particular energy. I do not believe that these substances, whether lifeless or living molecules, are always the same, but I think they are very numerous, as are the causes of inflammation generally; they may all have certain chemical peculiarities in common, as we might suppose from their similar action, although we know nothing about them, except this action; they also differ somewhat in their mode of action on this or that tissue; the absorbability of such substances may vary with the part of the body, and possibly, also, with the individual; but the large number of these injurious substances is, in fact, small as compared with the innumerable variety of organic substances generally.

*Febrile reaction* is usually greater from contused than from incised wounds; according to our view, this is because, from the decomposition, which is much more extensive in crushed than in incised parts, far more putrid matter enters the blood. If in any case the putrid matter is particularly intense, or very much of it is taken up (especially in diffuse septic inflammations), the fever assumes the character of so-called *putrid fever;* the state thus induced is called *septicemia;* we shall hereafter study it more closely. If the suppurative inflammation extends from the wound, there is a corresponding continued inflammatory or suppurative fever; this has the character of remittent fever with very steep curves and occasional exacerbations, mostly due to progress of the inflammation, or to circumstances that favor the reabsorption of pus. If we call the fever, that often, but not always, accompanies traumatic inflammation, simple *traumatic fever*, we may term the fever that occurs later "*secondary fever*," or "*suppurative fever.*" This may immediately succeed the traumatic fever, if the traumatic inflammation progresses regularly; but the traumatic fever may have ceased entirely, and the wound be already healing, and when new secondary inflammations, of which we have fully treated, attack the wound, they are accompanied by new suppurative fever; in short, inflammation and fever go parallel. Occasionally, indeed, the fever *appears* to precede the secondary inflammation, but this is probably because the first changes in the wound, which may be only slight, have escaped our observation. At all events, on every accession of fever that we detect, we should at once seek for the new point of inflammation, which may be the cause. I am far from asserting that it is necessary to measure the temperature in all cases of wounds; undoubtedly any experienced surgeon, accustomed to examine patients, would know the condition of his patient without measuring the temperature, just as an experienced practitioner may diagnose pneumonia without auscultation and percussion; but no one who understands the significance of bodily temperature doubts that its measurement may sometimes be a very important aid to diagnosis and prognosis. It is with it as with every other aid to observation; it is not difficult to detect a dull percussion-sound in the thorax where it should not exist; but the art and science of determining the significance of this dull percussion-sound in any given case must be learned; so, too, with measurement of temperature: for instance, we must learn whether a low temperature in any given case be of good or bad omen. I shall enter into more detail on this subject in the clinic.

Experience teaches that secondary fever is often more intense than primary traumatic fever. While it is most rare for the latter to begin with a chill (a slight chilliness after great loss of blood and severe concussion is not usually accompanied by high temperature), it is not at all so for a secondary fever to commence with severe "chill."

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We shall at once study this peculiar phenomenon more attentively Formerly the chill was always regarded as essentially dependent on blood-poisoning; if we now regard fever generally as due to intoxication, we must seek some special cause for the chill. Observation shows that the chill, which is always followed by fever and sweating, is always accompanied by rapid elevation of temperature. If we thermometrically examine the temperature of the blood of a patient with chill, we find it high and rapidly increasing, while the skin feels cool; the blood is driven from the cutaneous vessels to the internal organs. As already remarked, *Traube* considers this as the cause of the abnormal febrile elevation of temperature. We shall not discuss this at present; at all events, there is so great a difference between the air and the bodily temperature that the patient feels chilled. If we uncover a patient with fever, who lies wrapped up in bed and does not feel chilly, he at once begins to shiver. Man has a sort of conscious feeling for the state of equilibrium in which his bodily temperature stands to the surrounding air; if the latter be rapidly warmed, he at once feels warmer, if it be rapidly cooled, he at once feels cool, chilly. This trivial fact leads us to another observation. This sensitiveness for warmth and cold, this conscious feeling of change of temperature, varies with the individual; it may also be increased or blunted by the mode of life; some persons are always warm, others ever too cold, while for others the temperature of the air is comparatively a matter of indifference. The nervous system has much to do with this. Accurate studies of *Traube* and *Jochmann* have in fact shown that the nervous excitability of an individual has a great effect as to whether, in a rapid elevation of temperature of the blood, the change will be much perceived or not; hence that in torpid persons, in comatose conditions, chills do not so readily occur with fever, as they do in irritable persons already debilitated by long illness. I can only confirm this from my own observation. Although I have a general idea that, where there is sufficient irritability, rapid elevation of temperature and chill chiefly occur when a quantity of pyrogenous material enters the blood at once, still I cannot deny that the quality of the material is also important. We know nothing of this quality chemically, but we may conclude that it has varieties, because both the fever-symptoms and their duration often vary greatly, and that this does not solely depend on the peculiarities of the patient. According to my observations, in man reabsorption of pus and recent products of inflammation is more apt to induce chills than is absorption of putrid matter, which is perhaps more poisonous and dangerous. I do not wish to weary you with too many of these considerations, and so shall return to the subject in the section on general accidental traumatic and inflammatory diseases, which you may regard as a continuation of this study of fever. I will only remark here that both the septic and purulent primary and secondary inflammations, with their accompanying fever, may also occur from incised wounds, especially after extensive operations (as amputations and resections). We have considered this condition along with contused wounds, because it complicates them much more frequently than it does ordinary incised wounds.

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[Decomposition may begin in a contused wound within twenty-four hours after the injury; the tissue may have been crushed so that circulation is arrested and its substance infiltrated with blood and lymph; it may also contain foreign bodies, and ferments from the air enter it; hot weather may favor the decomposition, and even in a few hours the primary secretion may have an odor. If some of this secretion be injected into an animal it induces intense symptoms of local and general septic infection.

Progressive suppurative inflammation of cellular tissue, occurring especially in wounds of the hands and feet, may develop without the pus becoming foul; this is due to extension to the healthy tissue, the pus acting as an infector; wherever it enters the tissue it induces inflammation and suppuration, which may thus creep along the interspaces of the connective tissue between the tendons, etc.

In granulating, secondarily inflamed wounds the whole granulation layer, the interspaces in the connective tissue under it, many blood and lymph vessels, may be found filled with micrococci, apparently in rapid growth, and fungi active or quiescent may be found in the fluid secretion, even while it has no putrid odor; in fact, this is only apt to occur when the pus is confined in pockets.

When permanent germs have developed from these micrococci they prove quite resistant, and may be dried and kept for some time; then under favorable conditions, as in decomposing organic matter, they develop and multiply enormously and penetrate the tissue, inducing inflammation mechanically by their numbers and chemically by transporting phlogogenous material. To live and multiply, the fungi must supplant the albuminous bodies; this double action enables them to penetrate healthy granulations; dust-like micrococci, or irritant matter containing spores, reaching the granulating surface, may attack the life of the cells and destroy the surface.

Infecting matters may enter the economy by the air-passages; infectious diseases spread from substances entering the organism.

which act as poisons on the blood and the whole body; but we cannot say if the same morbid matters inducing the infectious diseases occurring especially in the wounded, may enter anywhere except by the wound.

If the inflammation be suppurative, it will keep up an inflammatory or suppurative fever, which has a remittent character, with steep curves and temporary exacerbations; this is due to progress of the inflammation, or circumstances favoring absorption of pus.

To recognize this fever, it is important to try the temperature at regular hours with the thermometer; this may give an early alarm to the surgeon of something going wrong in the wound; it is an aid both to diagnosis and prognosis.

In contused wounds we should care especially for free escape of secretion by drainage, and to prevent infection of the wound by anything that might excite decomposition, or at all events to limit this.

Let us suppose a recent contused wound with moderate destruction of the soft parts, extravasation of blood, and disturbance of circulation in the adjacent parts; the first care should be to cleanse it perfectly of foreign bodies, blood-clots, and tissue fluids, by passing over it a stream of a two-per-cent. solution of carbolic acid; of course any hæmorrhage is to be arrested; drainage-tubes should be applied, the wound again washed with a four-per-cent. solution of carbolic acid, and a *Lister's* dressing applied.

It is a disputed point whether sutures should be used in such wounds; if the contusion be considerable, we may not expect union by first intention, and accurate apposition of the edges would be useless or even injurious; but sutures may prevent wide gaping or excessive retraction of the flaps, which would retard union; hence deep sutures may be introduced to fix the flaps in place without accurately approximating them. If the wound be small and only the edges contused, the contused part may be cut off and united as in any incised wound.

Slight wounds, under *Lister's* dressing, run their course almost without reaction, the mortified tissue being gotten rid of by molecular disintegration and absorption. Gangrenous shreds do not appear, the crushed parts recover by restoration of the circulation or by plasmatic circulation, and entrance of wandering cells; this dead tissue is also consumed by the granulation tissue, which forms on the borders of the living, just as are portions of dead tissue introduced into the abdomens of animals. Under such circumstances suppuration may be slight or absent, there may be merely a muco secretion without cells, at first brownish-red, later yellowish, and some parts may unite by first intention.

If, in the contusion, there has been extensive crushing and tearing of the soft parts or openings into the joints or sheaths of the tendons, fractures of the bones, etc., frequently *Lister's* dressing does not answer the purpose, from the difficulty of cleansing the wound and later of preventing decomposition. Frequently such wounds are not recent when they come under treatment; they may be unclean from foreign bodies or applications of arnica, poultices, etc. In such cases it is well not to try Listerism, but to treat the wound open: cleanse it carefully, guard against retention of secretion, enlarging the skin-wound if necessary; if the soft parts are undermined, make counter-openings; cut off parts incapable of living, and wash the wound with a six- to eight-per-cent. solution of chloride of zinc, which is disinfectant and slightly cauterant; this gives the blood a red hue and the tissue a grayish-white color. Apply drainage-tubes and fill the pockets with some charpie or prepared cotton soaked in a disinfectant solution. This prevents superficial adhesions as well as retention of the secretions. To the wound you apply plenty of charpie, and cover the whole, far beyond the contusion, with compresses also soaked in disinfectant (such as acetate of alumina), then gutta-percha paper and a snug bandage.

The dressing should be changed daily, and, if there is much discharge, twice daily—else the disinfectant will not suffice. This dressing also supplies *moist warmth*, which has a favorable effect on the previously-disturbed circulation; redness and œdematous swelling of the edges of the wound diminish, and granulations develop. Dressings with *Burow's* fluid are cheaper than *Lister's* dressing, and less irritating than carbolic solutions.

Each time the wound is dressed it should be carefully washed and examined for collections of pus. If the escape of matter be anywhere impeded the opening should be enlarged, or a counter opening made by passing a silver catheter or a flexible metal sound to the bottom of the sinus, then cutting down on it. During suppuration the wound should be thus dressed; when the cavity is filled up cicatrization may be hastened by some irritating salve, as of red precipitate, iodoform, etc.

After injuries by machinery, railroad accidents, etc., there may be such crushing of the tissue as to cause primary mortification of the skin and soft parts. The muscles are crushed to a pulp, and with the extravasations, etc., may form a flaccid bladder communicating outwardly by a small wound in the skin; the skin being very resistent to crushing force, as is observable on using *Chassaignac's* *écraseur*, it may be so compressed as to be bloodless and leathery, but it is difficult to divide it completely; where the crushing has been extensive, but the opening in the skin is small, this should be enlarged to expose the cavity for treatment; in such cases open treatment is insufficient, from the danger of decomposition at some point of the dead tissue exposed to moisture at the heat of the body. Here permanent antiseptic irrigation may be resorted to, by dressing as above described, with charpie in the wound and covering with compresses, only not applying gutta-percha, but suspending an *Esmarch's* irrigator so that *Burow's* solution may drop on the moist compresses and keep them wet; the injured part being placed on a rubber cloth so that the superfluous liquid may run from it into a vessel under the bed; constant renewal of the disinfecting fluid in the dressing is thus secured. This plan is suited for severe injuries of the hand and fingers. If the wound be extensive, deep, and irregular, it is well to lead the irrigation through the drainage-tubes to the bottom of the wound, for which purpose you may let one or two of the chief tubes project through the dressing and connect with the irrigator; the fluid may escape through another tube or through the wound. When the apparatus is in good working order, it requires little further attention. The effect is very good, the necrosed parts being quickly detached and healthy granulations developed. Irrigation also acts by abstracting heat and relieving pain, and is very acceptable to the patient, except when the bed is accidentally wet. It is best suited for applying immediately after the injury; when the wound has cleaned and granulated, this dressing may be changed for simple moist dressing.

The results from this treatment are very favorable as compared with those by old methods. Besides the slight local reaction there is absence of severe general infectious disease if the irrigation be applied early. The temperature may rise high, but other signs of fever are absent.

If, from improper treatment, decomposition and putrefaction have started, if the edges of the wound and the soft parts are swollen, infiltrated with serum, and symptoms of general infection are present, if amputation is not directly indicated, treatment should be preceded by energetic disinfection and evacuation of the septic matter. This is to be done by opening the wound to its full extent, washing with a four-per-cent. solution of carbolic acid, and rubbing with sponges; the mortified tissues are removed, and where the skin is gangrenous it is to be divided to where it is healthy; pockets are to be opened; numerous deep punctures should be made in the infiltrated tissue to let the serum escape, according to *Volk-*

*mann's* plan; then the whole should be washed and rubbed with a four-per-cent. solution of carbolic acid, and *Burow's* solution applied on compresses and irrigation started.

Even when contused wounds have begun to suppurate decomposition may occur from retained secretion. This is shown by redness and swelling of the edges, or by sudden severe pain and fever; on examination you find a sensitive spot, on pressing which thin fetid pus escapes from some part of the wound. Often there are several such points, or the suppuration is in the sheath of a tendon or between muscles, and we have a long sinus. In such cases counter-openings are indicated, or even laying open the whole fistulous tract, and scraping with a sharp spoon the relaxed granulations, and cauterizing the surface with a concentrated solution of chloride of zinc. In such cases also the acetate of alumina is the best dressing, but it should be applied at least once daily.

If the inflammation be diphtheritic in character, with hard infiltration, deep incisions should be made to prevent gangrene, and moist warmth applied (compresses moistened with hot *Burow's* solution) to break down the infiltration and hasten suppuration.

This is the only reason for using poultices, which were formerly so common, because they retain heat better than hot-water compresses. They should be banished from the hospital as a dirty dressing with many objections; in private practice, where their preparation may be watched, they may sometimes be employed; the warmth is agreeable to the patient.

We have incidentally mentioned quiet and position in the treatment; in this class of injuries it is the more important, as excitement of the vascular system induces congestion of the injured parts and is injurious. Hence not only should the injured part be kept motionless, but the whole body should be kept quiet, the patient remaining in bed and avoiding all motion. Mental quiet is also most desirable.

When possible, the injured part should be elevated. Gravity has some effect on the circulation, as you may see by letting your arm hang relaxed for five minutes, when the hand will feel heavy and the veins on its back be filled; but if you hold it up the hand grows white and smaller. When feeble persons lie in bed their faces look fuller than after they have been up some time. For injuries of the hand *Volkmann* has recommended vertical suspension of the arm; the same treatment may be applied for inflammations of the leg. In this position pain is speedily lessened; for deep-seated inflammations it is less useful.

Another remedy is regular compression by a roller bandage, which may be applied over *Lister's* dressing; this improves the circulation and prevents further swelling; rubber bandages rather loosely applied may replace muslin for this purpose; this also tends to relieve pain.

The most important rules for avoiding accidental complications of wounds are prophylactic, and consist in rigid antisepsis, not only in treating the wound, but in the whole care of the patient. The surgeon should see to the disinfection of the furniture and everything in the room, to the ventilation and cleanliness, and to the removal of old dressings, etc. If attention be merely given to the wound, results will be unsatisfactory. Instruments, sponges, dressings, etc., should be kept constantly disinfected. For hospitals new sponges should be cleaned, disinfected, and kept in a closed glass vessel in a four-per-cent. solution of carbolic acid, which should be changed every week, and be washed again in carbolized water before using. Catgut and silk should be boiled for an hour in a four-per-cent. solution of carbolic acid. Carbolized gauze loses its strength after a time, hence should be prepared fresh frequently. Gutta-percha paper, the instruments, etc., should be washed with carbolized water before using. Nurses should be trained to consider antisepsis, as well as cleanliness, second nature.

*Spencer Wells*, the ovariotomist, demanded that physicians wishing to see his operations should not have visited a dissecting or autopsy room for some days previously, hoping thus to escape infection from their clothing. Danger from this cause may probably be avoided by washing in disinfectant solutions on leaving the dissecting or autopsy room. It is proper to remove the outer clothing before entering the sick-room, as infecting germs readily adhere to rough cloth. In the hospital it is well to wear a linen blouse with the sleeves tight at the wrist, and when this is soiled put on a fresh one; this is not to protect the clothes of the surgeon, but to guard against infection.

This advice is, of course, applicable to any kind of wound, whether from accident or operation. You cannot be too exact in observing the above rules; they are applicable not only to hospitals, but just as much so to private practice.

Cooling drinks and medicines and proper diet should be prescribed: if a remittent fever occurs, rendering the patient dull and sleepless, quinine and opium may be beneficial. If the fever be slight, give nothing; if fever increases toward evening, two five-grain doses of quinine, with bicarbonate of soda, may be given, and the eighth of a grain of morphine at bedtime; on cessation of the fever this may be discontinued.

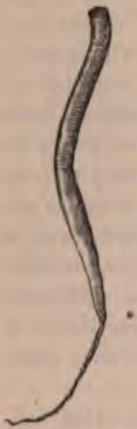
Now a few words about *lacerated wounds*, by which we mean those where there is a division of the soft parts and the skin or of the latter alone, characterized chiefly by the irregular, uneven edges of the wound; different tissues having different cohesiveness and elasticity, the same force tears some more readily than others, and some retract more than others. At the time of injury the pain is severe, but soon ceases, as the nerves have been torn and fail to conduct. Hæmorrhage is usually slight, even if large arteries have been ruptured; this is due to the mode of injury. If an artery be stretched lengthwise, the intima and media tear first, they roll up and obstruct the calibre; if the traction continue, the tougher adventitia is drawn out into a solid string, which finally ruptures in the middle, leaving two conical ends, plugged within by a mechanical obstruction about which blood quickly coagulates. Hæmorrhages from ruptured veins may be much freer, because their adventitia is much weaker, and is apt to be torn with the inner membranes.

Microscopic examination of a lacerated wound shows that it is almost always the intercellular substance, not the cells, that is torn. The destruction is less than in contused wounds, hence the prognosis is more favorable; the wound being more exposed, there is less fear of injury of deep tissues, and we see the full extent of injury. Healing by first intention is not rare, as the edges of the wound have not been crushed, and, although many blood-vessels may have been ruptured, circumstances are favorable for plasmatic circulation.]

But ruptures are not always exposed; there are also *subcutaneous ruptures* of muscles, tendons, or even of bones, without there having been any contusion. A person wishes to leap a ditch, and makes a start, but fails in his attempt; he falls, and feels a severe pain in one leg, and limps on it. On examination, just above the heel (the tuberositas calcanei), we find a depression in which the thumb may be laid; the motions of the foot are imperfect, especially extension. What has happened? The tendo Achillis has been torn from the calcaneus by the great muscular action. The same thing occurs with the tendon of the quadriceps femoris, which is attached to the patella, with the patella itself, which may be torn in two, with the ligamentum patellæ, with the triceps brachii, which may be torn from the olecranon, and generally carries a piece of the latter along with it. Here you have a few examples of such subcutaneous ruptures of tendons; I have seen subcutaneous rupture of the rectus abdominis, of the vastus externis cruris, and other muscles. These simple subcutaneous ruptures of muscles are not serious injuries;

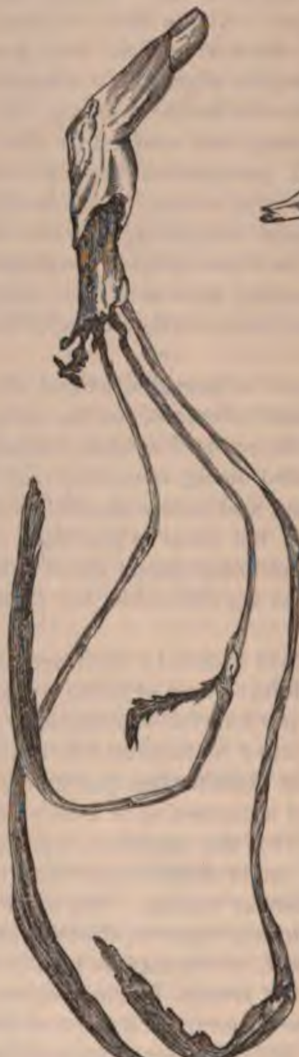
they are readily recognized by the disturbance of function, by the depression, which may be seen and still better felt, which at

FIG. 40.



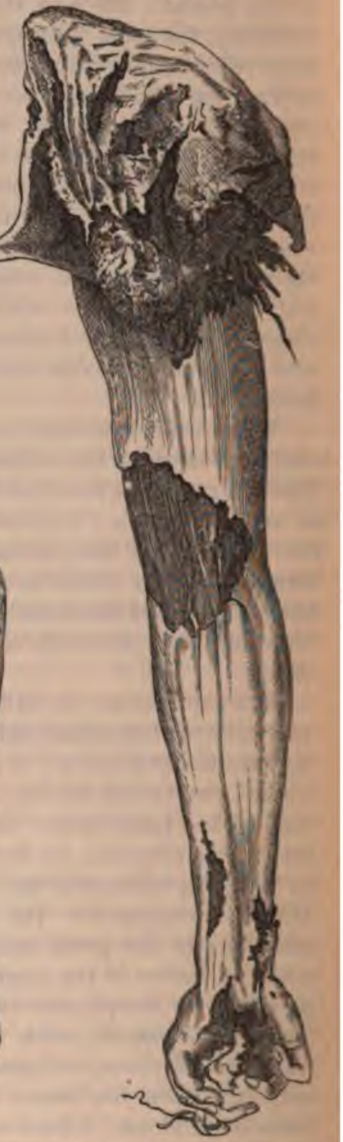
Central end of a torn brachial artery.

FIG. 41.



Torn-out middle finger, with all its tendons.

FIG. 42.



Arm torn out, with scapula and clavicle.

once occurs, but subsequently is masked by the effused blood. The treatment is simple: rest of the part, placing it so that the ruptured ends may be brought in contact by relaxation of the muscle, cold compresses, lead-water lotions for several days; after eight or ten days the patient can generally rise without pain; at first there is a connective-tissue intermediate substance, which soon condenses so much, by shortening and atrophy, that a firm tendinous cicatrix forms; the course is just the same as in subcutaneous division of tendons, of which we shall speak in the chapter on deformities.

Functional disturbances of any considerable amount rarely remain; occasionally there is some weakness of the extremity and loss of delicate movements, especially in the hand.

For such subcutaneous rupture of muscles and tendons to be caused by contusion, the crushing force would have to be very great; such a contusion would probably run a bad course; extensive suppurations and necroses of tendons might be expected. Here, again, you see how varied may be the course of injuries apparently the same, according to the mode of their origin. In injuries by machinery there is often such a wonderful combination of crushing, twisting, and lacerating, that even with great experience it is very difficult to give any accurate prognosis of their course. The favorable course of cases, where small or even large portions of a limb (as the hand) are torn off, is especially worthy of mention. I have seen two cases where fingers were torn off; I will briefly narrate one of them: a mason was employed on a scaffolding, and suddenly felt it giving way under him; from the roof of the house against which the scaffold rested there hung a loop; the falling man grasped this, but only succeeded in getting the middle finger of the right hand through the loop; he hung a moment and then fell to the ground. Fortunately, the height was not great, and he was not injured, but the middle finger of the right hand was gone; it was torn out at the joint between the first phalanx and the metacarpal bone, and it still hung in the loop. The two tendons of the flexors and that of the extensor remained attached to the finger; they had been torn off just at the insertion of the muscles; the man dried his finger with the tendons, and subsequently carried it in his purse as a memento of the circumstance. I saw a similar case in the clinic at Zurich (Fig. 41). Cure resulted without much inflammation of the forearm, and actually no treatment was required. In Zurich I saw two cases where the hand was torn out; in one case there was enough skin remaining to leave the healing to itself, in the other case an amputation of the forearm was necessary. Both cases terminated favorably. In war it is not very rare for arms and legs to be torn from their sockets by large cannon-balls. I have also seen a case where a boy fourteen years old had the right arm with the scapula and clavicle so torn from the thorax, by a wheel of machinery, that it was only attached at the shoulder by a strip of skin two inches wide (Fig. 42). The axillary artery did not bleed a drop; both ends were closed by torsion (Fig. 40). The unfortunate fellow died soon after the injury. Tearing out of entire extremities is usually quickly fatal.

# CHAPTER V.

## *SIMPLE FRACTURES OF BONES.*

### LECTURE XIV.

Causes, Different Varieties of Fractures.—Symptoms, Diagnosis.—Course and External Symptoms.—Anatomy of Healing, Formation of Callus.—Source of the Inflammatory Osseous New Formation.—Histology.

GENTLEMEN: Hitherto we have been exclusively occupied with injuries of the soft parts; it is time to consider the bones. You will find that the processes that Nature excites for the restoration of the parts are essentially the same that you already know; but the circumstances are more complicated, and can only be fully understood when you are perfectly acquainted with the mode of healing in the soft parts. Every person knows that bones may be broken, and again be firmly united; this can only be done by bony tissue, as you will at once see; hence it follows that new bony substance must be formed; the cicatrix in bone is usually bone; a very important fact, for, if this were not the case, if the broken ends only grew together by connective tissue, as divided muscles do, the long bones particularly would not be united firmly enough to support the body, and after the simplest fractures many men would be cripples for life. Still, before following the process of the healing of bones to its more minute details, a study that has always been pursued with great zeal by surgeons, I must tell you something about the origin and symptoms of simple fractures; I say "simple or subcutaneous fractures" in contradistinction to those accompanied by wounds of the soft parts.

Man may even come into the world with broken bones: the bones of the fœtus may be broken, while in the uterus, by abnormal contractions of that organ, or by blows or kicks on the pregnant abdomen, and such intra-uterine fractures generally heal with considerable dislocation; as we shall see in other instances, the *vis medicatrix naturæ* is a better physician than surgeon. Of course, fractures of the bones may occur at any age, but they are most frequent between the ages of twenty-five and sixty years, for the following reasons: The bones of children are still pliable, and hence do not break so easily; if a child falls, it does not fall heavily. Old people have, as is commonly remarked, brittle, friable bones; or, anatomically expressed, in old age the medullary cavity grows larger, the cortical substance thinner; but old persons are less in danger of fractures of the bones, because their lack of strength prevents their doing hard and dangerous work. It is during the age when men are most exposed to hard work that injuries generally and fractures especially are most liable to occur. The less frequency of fractures among women is due to the variety of their occupation. It is also due entirely to external circumstances that the long bones of the extremities, especially of the right side, break more frequently than those of the trunk. It is evident that diseased bones, which are already weak, break more easily than healthy ones; hence certain diseases of the bones greatly predispose to fractures, especially the so-called English disease, "rickets," which is due to deficient deposit of lime-salts in the bones, and only occurs in children; also softening of the bones or "osteomalacia," which depends on abnormal dilatation of the medullary cavity, and thinning of the cortical substance, and which is, to a great extent, accompanied by a "fragilitas ossium," and even by total softness and flexibility of the bones.

As special causes of fractures, we have the two following: 1. The action of external forces, the most frequent cause; this action may vary in the following ways: the force—for instance, a blow or kick—meets the bone *directly*, so that it is crushed or broken; or the bone, especially a long bone, is bent more than its elasticity permits, and breaks like a stick that is bent too much; here the force acts *indirectly* on the point of fracture. In the mechanism of the latter variety, instead of the single hollow bone, you may consider a whole extremity or the entire spinal column as a stick, flexible to a certain extent, and on this supposition found your idea of the indirect action of the force. Let us have a couple of examples to explain this: If a heavy body falls on a forearm at rest, the bones are broken by direct force; if a person falls on the shoulder, and the clavicle is broken obliquely through the middle, this is the result of indirect force. In both cases there is usually contusion of the soft parts; but in the latter case it is more or less removed from the point of fracture; in the former at that point, which evidently is to be regarded as less favorable.[7]

2. Muscular action may, though rarely, be the cause of fracture. As I already indicated, when speaking of the subcutaneous rupture of

muscles, the patella, the olecranon, and part of the calcaneus also, may be torn off by muscular action, that is, obliquely fractured.

The way in which the bones break under these varied applications of force varies, but some types have been formed that you should know. First, we distinguish complete and incomplete fractures. *Incomplete fractures* are again subdivided into *fissures*, i. e., clefts, cracks; they are most frequent in the flat bones, but occur also in the long bones, especially as longitudinal fissures accompanying other fractures; the cleft may gape or appear simply as a crack in glass. *Infraction*, or bending, is a partial fracture, which, as a rule, only occurs in very elastic, soft bones, and especially in rachitic children; you may best imitate this fracture by bending a quill till its concave side breaks in. In children, such infractions of the clavicle are not rare. What we mean by *splintering* is evident; the most frequent causes are machine-cutters, sabre-strokes, etc. Lastly, the bone may be perforated without entire solution of continuity, as by a punctured wound through the scapula, or a clean shot through the head of the humerus. The latter variety of injury is called a *perforated fracture.*

*Complete fractures* are subdivided into *transverse, oblique, longitudinal, dentate, simple,* or *multiple* fractures of the same bone, *comminuted;* all of these expressions explain themselves. Lastly, we must mention that persons as old as twenty years may also have a solution of continuity in the epiphyseal cartilages, although this is rare, and the long bones break more readily at some other point.

Frequently it is easy to recognize that a bone is broken, and a non-professional person may make the diagnosis with certainty; in other cases the diagnosis may be very difficult, and occasionally can only be a probable one.

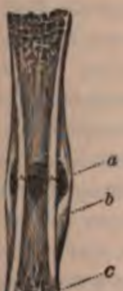
Let us take up the symptoms one after another. First, accustom yourself to examine every injured part accurately, and compare it with healthy parts; this is particularly important in the extremities. You may not unfrequently know what the injury is by simple observation of the injured extremity. You ask the patient how it happened, having him undressed meantime, or, if this be painful, have his clothes cut off, that you may accurately examine the injured part. The manner and severity of the injury, the weight of any body that has fallen on the part, may indicate about what you have to expect. If you find the extremity crooked, the thigh bent outward, for instance, and swollen, if suggillations appear under the skin, if the patient cannot move the extremity without great pain, you may with certainty decide on a fracture; here you need no further examination to decide on the simple fact of a fracture, it is not necessary to put the patient to any pain on this account; you have only to examine with the hands to find how and where the fracture runs; this is less necessary, on account of determining the treatment, than to be able to decide whether and how recovery will result. In this case you have made the diagnosis at a glance, and in surgical practice it will often be easy for you to recognize very quickly the true state of affairs, when you are accustomed to use your eyes thoughtfully, and when you have acquired a certain habit in judging of normal forms of the body. Nevertheless, you should know perfectly how you arrived at this sudden diagnosis. The first point was the mode of the injury, then the deformity; the latter is caused by two or more pieces of bone (fragments) having been displaced. This dislocation of the fragments is due partly to the injury itself (they are driven in the direction that they maintain, from the bending of the bone), partly to the muscular action which no longer affects the entire bone, but only a part; the muscles are excited to contraction, partly by the pain from the injury, partly by the pointed ends of the bone; for instance, the upper portion of a fractured thigh-bone is elevated by the flexors, the lower portion is drawn up near or behind the upper fragment by other muscles, and thus the thigh is shortened and deformed. The *swelling* is caused by the effusion of blood (we speak here of a fracture that has just occurred); the blood comes chiefly from the medullary cavity of the bone, and also from the vessels of the surrounding soft parts which have been crushed or torn by the ends of the bone; it looks bluish through the skin, if it works up to the skin, as it gradually does. The patient can only move the extremity with great pain; the cause of this *disturbance of function* is evident, we need waste no words on it. If we examine each of the above symptoms separately, none of them, either the mode of injury, the deformity, swelling, effusion of blood, or functional disturbance, will alone be evidence of a fracture, but the combination is very decisive; and you will often have to make such a diagnosis in practice. But all these symptoms may be absent when there is fracture. If there has been an injury, and none of the above symptoms are well developed, or only one or other of them distinctly exists, manual examination must aid us. What will you feel with your hands? You should learn this thoroughly at once. I so often see practitioners feel about the injured part for a long time with both hands, causing the patients unspeakable pain, and after all finding out nothing by their examination. By the touch you may perceive three things in fractures: 1. *Abnormal mobility*, the only pathognomonic sign of fracture; 2. You may often detect the course of the fracture, and often whether there are more than two fragments; 3. By moving the fragments you will often experience a rubbing and cracking of the fragments against each other, the so-called "*crepita* *tion*"—strictly to crepitate means to crackle; this is a sound, and still we say, we feel crepitation; it is no use to object to this; this is an abuse of the word, which has so gone into practice, however, that it cannot be rooted out, and every one knows what it means. An educated touch usually feels at once all that can be detected by the touch; hence it is unnecessary to make the patient suffer long under this examination. Crepitation may be absent or very indistinct; of course, it only exists when the fragments can be moved, and when they are quite near each other; if they be considerably displaced laterally or be drawn far apart by muscular contraction, or if there be blood between the fragments, no crepitation can be felt, and it is often difficult to detect when the bones lie deep. Hence, if we detect no crepitation, this, in opposition to all the other symptoms, does not prove that there is no fracture. Still, even where there is crepitation, you may mistake its origin; you may have a feeling of friction under other circumstances; for instance, the compression of blood coagula or fibrinous exudations may give a feeling of crepitation; this soft crepitation, which is analogous to pleuritic friction, you should not and will not mistake for bony crepitus after some experience in examination; when opportunity offers, I shall hereafter call your attention to other soft friction-sounds which occur especially in the shoulder-joint in children and old persons. For experienced surgeons, in certain fractures severe pain at a fixed point is enough for a correct diagnosis, especially as in contusions the pain on grasping the bone is mostly diffuse, and rarely so severe as in fracture. If we are examining an extremity, it is best to seize it with both hands at the suspected point, and attempt motion here; this manipulation should be firm, but not rough, of course. I must add something about the dislocation of the fragments; this may vary, but the displacements may be divided in various classes, which from time immemorial have had certain technical designations, which are still used, and which consequently must be explained. Simple lateral displacement is called *dislocatio ad latus;* if the fragments form an angle like a half-broken stick, it is called *dislocatio ad axin.* If a fragment be rotated more or less on its axis, we call it *dislocatio ad peripheriam;* if the broken ends be shoved past each other vertically, it is a *dislocatio ad longitudinem.* The expressions are short and distinctive, and easily remembered, especially if you represent to yourselves the displacements by diagrams.

We now pass to a description of the course of healing of a fracture. You will rarely have the opportunity of seeing what happens when no bandage is applied, as the patient generally sends early for a surgeon. But occasionally the laity undervalue the importance of the injury; several days pass before the pain and duration of the affection at last cause the patient to apply to a surgeon. In such cases, besides the symptoms of fracture already given, you find great œdema, and in some few cases inflammatory redness of the skin about the point of fracture; under such circumstances the examination may be very difficult; occasionally the swelling is so considerable that an exact diagnosis as to the course and variety of the fracture is out of the question. Hence the earlier we see a fracture the better. The subsequent external changes at the point of fracture may best be studied on bones that lie superficially, and which cannot be surrounded with a bandage, as on fracture of the clavicle. After seven to nine days, the inflammatory œdematous swelling of the skin has subsided, the extravasated blood has run through its discolorations and goes on to reabsorption, and a firm, immovable, hard tumor lies around the point of fracture; this is larger or smaller according to the dislocation of the fragments; it is, as it were, poured around the fragments, and in the course of eight days becomes as hard as cartilage; this is called *callus*. Pressure on it (the fragments can with difficulty be felt through it) is painful, though less so than previously; subsequently the callus becomes absolutely firm, the broken ends are no longer movable, the fracture may be regarded as healed; for the clavicle this requires three weeks, in smaller bones a shorter, and in larger ones a much longer time. But this does not end the external changes; the callus does not remain as thick as it was; for months or years it grows thinner, and, if there was no dislocation of the fragments, after a time no trace of the fracture will remain; if there was a dislocation that could not be reduced by treatment, the ends of the bone unite obliquely and after absorption of the callus the bone remains crooked.

To find out the changes that take place in the deeper parts, how the fractured ends unite, we try experiments on animals. We make artificial fractures on dogs or rabbits, apply a dressing, kill the animals at various stages, and then examine the fracture; we may thus obtain a perfect representation of the process. These experiments have been made innumerable times. The results have always been essentially the same; but, if we speak of rabbits alone, there are certain variations which, as proved by numerous experiments, depend on the amount of dislocation and of extravasation of blood. Hence, before showing you a series of such preparations, I must give you the result of these investigations, and exemplify them by a few diagrams; then you will hereafter readily understand the slight modifications.

We shall first confine ourselves to what we can see with the naked eye and a lens. If you examine a rabbit's leg three or four days after the fracture, and, while it is firmly held in a vice, saw the bone longitudinally, you find the following: the soft parts about the fracture are swollen and elastic; the muscles and subcutaneous cellular tissue look fatty; the swollen soft parts form a spindle-shaped, not very thick tumor about the seat of fracture. About the broken ends we find some dark extravasated blood, and the medullary cavity at the same point is somewhat infiltrated with blood. The amount of this escaped blood varies, being sometimes very slight, again considerable. At the point of fracture the periosteum may be readily recognized, and is intimately connected with the other swollen soft parts (which are the seat of plastic infiltration). Occasionally it is somewhat detached from the bone at the point of fracture. The whole thing looks about as follows (Fig. 43):

Fig. 43.



Longitudinal section of a fracture of a rabbit's bone, four days old; *a*, extravasated blood; *b*, swollen soft parts external callus; *c*, periosteum.

Fig. 44.

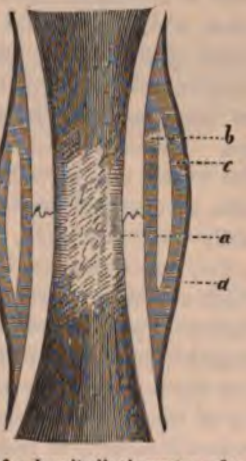
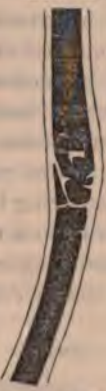


Diagram of a longitudinal section of a fifteen-day-old fracture of a long bone; *a*, internal callus; *b*, inner, *c*, outer layer of ossification of the external callus; *d*, new periosteum. The dimensions of the callus, in proportion to the entire lack of dislocation of the fragments, are represented as far too great, but this facilitates the preliminary understanding of the case.

If we now examine a fracture in a rabbit after ten or twelve days, we find that the extravasation has either entirely disappeared, or that only a slight amount remains. I will not raise the question as to whether it has been entirely reabsorbed, or has partly organized to callus. The spindle-shaped swelling of the soft parts has mostly the appearance and consistence of cartilage, and has also the same microscopical characteristics; in the medullary cavity also we find young cartilage formations in the vicinity of the fracture. The broken bone sticks in this cartilage as if the two fragments had been dipped in sealing-wax and stuck together; the periosteum is still tolerably distinct in the cartilaginous mass, but it is swollen, and its contours are indistinct. Although there are traces of ossification even now, they do not become very decided or evident to the naked eye for some days (perhaps the fourteenth to the twentieth day after the fracture). Then we see the following (Fig. 44):

In the vicinity of the fracture there is young soft bone: 1. In the medullary cavity (*a*). 2. Immediately on the cortical layer (*b*), and some distance up and down beneath the periosteum, which has disappeared in the whole spindle-shaped callus tumor. 3. In the periphery of the callus, which is still mostly cartilaginous (*c*). The periosteum which previously lay within the callus has now disappeared; in its place a thickened layer of tissue has formed on the outside of the callus, which represents the periosteum (*d*). The young bone-substance is soft, white, and in it we may see a kind of structure; for small parallel pieces of bone, corresponding to the transverse axis of the bone, may be distinctly seen, especially on examination with a lens. The cartilaginous callus formed from the surrounding soft parts, into which the periosteum also has been partly transformed, now forms an enclosed whole, and ossifies entirely, partly from without (*c*), partly from within (*b*), till finally the ends of the bone stick in bony, as they previously did in the cartilaginous callus. This bony callus, which consists entirely of spongy bone-substance, is called by ***Dupuytren*** "*provisional callus.*" As it is completed, the bone is usually firm enough to be again capable of function; but the callus does not remain in its present condition any more than a recent cicatrix of the soft parts does. A series of changes occurs in it in the course of months or years, for up to this point you may still compare the union to that by sealing-wax, which is not a true organic union. So far the firm cortical substance is only united by loose young bone-substance; the medullary cavity is plugged with bone; the healing is not yet solid; Nature does far more. We shall now study the subsequent changes; they are confined to the spongy substance of the callus. At a certain time this ceases to increase, and then changes, by reabsorption of the bony substance that has formed in the medullary cavity (Fig. 45), and by the disappearance of a great part of the external callus. Meantime, formation of new bone has commenced between the fractured cortical layers, so that this has become solid by the time the external and internal callus disappears. This connecting bony substance between the fragments gradually increases in density, to such an extent that it becomes as hard as the bone in the normal cortical substance. In case there has been little or no displacement of the fragments, the bone is thus so fully restored that we can no longer determine the point of fracture, either on the living person or the anatomical preparation.

FIG. 45.



Longitudinal section of a fractured bone from a rabbit, after twenty-four weeks. Progressive reabsorption of the callus. Restoration of the medullary cavity, natural size; after *Gurlt.*

The above changes occur in a long bone of a rabbit, where there has been little displacement, in about twenty-six or twenty-eight weeks, but in the long bones of man last much longer, so far as we can judge from preparations that we accidentally have the opportunity of examining.

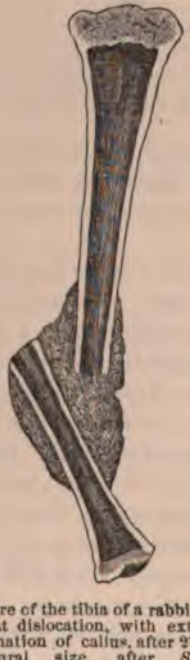
The entire process, so excellently contrived by Nature, is essentially the same as what we observe in the normal development of the long bones; for there, too, the same reabsorption and condensation take place in the medullary canal and the cortical layers of the long bones, as we have just studied in formation of callus. Except the regeneration of nerves, no such complete restoration of a destroyed part takes place in any other part of the human body as we have seen occurs in the bones.

I must still add a few remarks about the healing of flat and spongy bones. In the case of the first, which we see most frequently in the healing of fissures of the cranial bones, the development of provisional callus is very slight, and occasionally appears to be entirely wanting. In the scapula, where dislocation of small, or half or wholly detached fragments is more apt to occur, external callus forms more readily, although even here it never becomes very thick. On the union of spongy bones, too, in which, as a rule, there is also but little dislocation, there is less development of external callus than in the long bones; while, on the other hand, the cavities of the spongy substance in the immediate vicinity of the fracture are filled with bony substance, of which part, at least, subsequently disappears.

As may readily be imagined, the conditions will be somewhat more complicated when the ends of the bone are much dislocated, or when fragments are entirely broken off and displaced. In such cases there is such a rich development of callus, partly from the entire surface of the dislocated fragments and from the medullary cavity, and partly in the soft parts between the fragments, that for some distance all the fragments are embedded in a bony mass, and organically glued together. The larger the circle of irritation from the dislocated fragments, the more extensive the formative reaction.

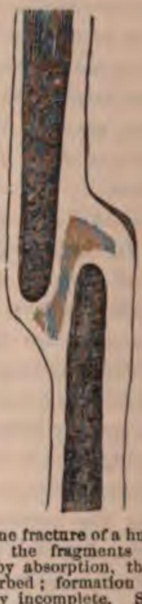
In man we most frequently have the opportunity of seeing callus formation in greatly dislocated fractures of the clavicle, where it is very evident that the extent of the new formation of bony substance is directly proportional to the amount of dislocation. You may readily understand how, in this way, with extensive formation of neoplastic bone-substance, there may be perfect firmness, even with great deformity at the point of fracture. Still, one would hardly believe, without satisfying himself on the point, from preparations, that with time, even in such cases, Nature has the power of restoring, not only the outward shape of the bone (except the curvature and rotation), but also the medullary cavity, by reabsorption and condensation. Numbers of points, nodules, inequalities and roughnesses of all sorts, that are formed on the young callus in recent cases, so disappear in the course of months and years, that in their place there is only left some dense, compact, cortical substance.

FIG. 46.



Fracture of the tibia of a rabbit, with great dislocation, with extensive formation of callus, after 27 days. Natural size, after *Skutsch*. (*Gurlt's* Fractures, vol. i., p. 270.)

FIG. 47.



Old united oblique fracture of a human tibia; the ends of the fragments have been rounded off by absorption, the external callus reabsorbed; formation of the medullary cavity incomplete. Size diminished. *Gurlt*, l. c., p. 287.

It will now be interesting to investigate the true origin of the newly-formed bony substance; is it produced by the bone itself, by the periosteum, by the surrounding soft parts, or is the extravasated blood transformed into bone, as was believed by old observers? Must formation of cartilage always precede that of bone, or is this unnecessary? These questions have received various answers, till quite recently. To the periosteum, especially, great power of producing bone has at one time been ascribed, at another denied. In what follows, I will briefly give you the results of my investigations on this subject.

The new formation that results from the fracture occurs in the medulla and Haversian canals of the bone, in the periosteum, and infiltrated in the adjacent muscles and tendons; possibly the extravasated blood may also have something, but very little, to do with the formation of the callus; a large extravasation is disturbing here, as in healing of wounds of the soft parts, for part of it must be organized, while the remainder is absorbed. The inflammatory new formation here, also, at first consists of small round cells, which increase greatly in number, and infiltrate the tissues mentioned, and then almost take their place. Before following the fate of this cell-formation further, I must briefly consider its course in the Haversian canals. The cell-infiltration in the connective tissue of the medullary cavity offers nothing peculiar, except that the fat-cells of the medulla disappear in the mass as the wandering cells take possession of the territory. Suppose the following figure (Fig. 48) to represent the surface, or the fractured surface, of a bone on which, as you know, the Haversian canals open; in these canals lie blood-vessels, surrounded by some connective tissue.

If this bony surface be in the vicinity of a fracture, numerous

FIG. 48.

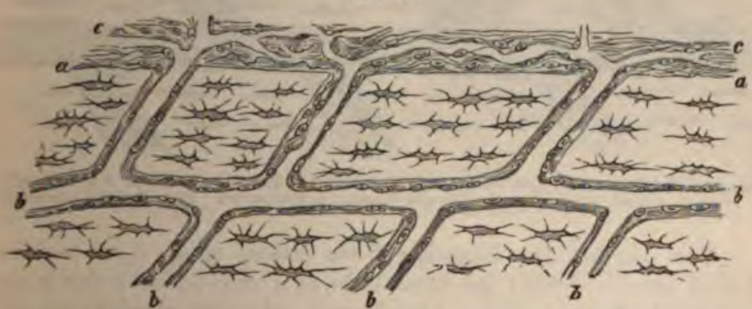


Diagram of a longitudinal section through the cortical substance of a long bone. *a*, surface; *b*, Haversian canals, with blood-vessels and connective tissue; *c*, periosteum. Magnified 400 diameters.

cells first come between the connective tissue in the Haversian canals; should this cell-infiltration be very rapid, it would entirely compress the blood-vessels, and cause the death of the bone, a process which we shall hereafter learn. But, if the cell-increase in these canals goes on slowly, their walls are gradually absorbed, as it would appear, by the inflammatory new formation itself; the canals are dilated, the cells fill them, and at the same time the blood-vessels increase by forming loops.

From the observations of *Cohnheim*, we must suppose that in inflammation of bone, also, the young cells in the Haversian canals are not newly formed, but are white blood-cells escaped from the vessels. This has no effect on the subsequent course.

Now, let us turn to the changes of form that we observe in the osseous tissue. As the connective tissue of the osseous canals is continuous, both with the periosteum and medulla, the cell-infiltration into the bone, periosteum, and medulla, is also continuous. The cause of the atrophy of bone along the walls of the Haversian canals, which takes place in this, as in most other new formations in the bone, is difficult to explain; the disappearance of the connective tissue and muscular substance, as well as of other soft structures, when the inflammatory new formation occurs in them, is less strange; but it is truly remarkable that hard bony substance should thus be dissolved. This process might be represented by the following diagram (Fig. 49):

FIG. 49.

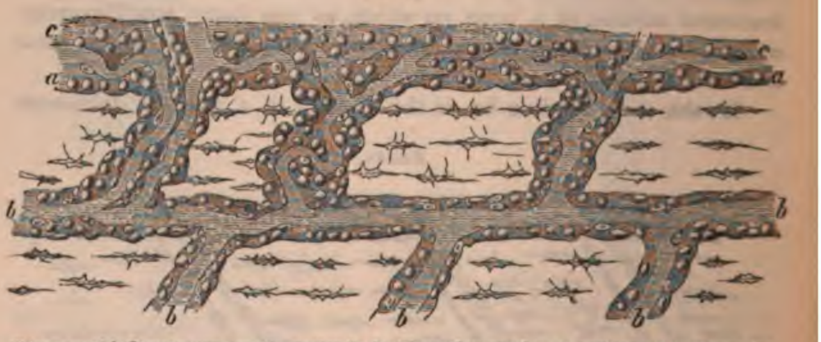


Diagram of inflammatory new formation in the Haversian canals. *a*, surface; *b b*, Haversian canals, dilated, filled with cells and new vessels; *c*, periosteum. Magnified 400 diameters.

You see that the dilatation of the osseous canals is not regular, but of uneven widths; the bone looks as if gnawed out; this is not necessarily so, the atrophy of the bone may be more regular; according to my idea, these irregularities result from the collection of cells in groups, or from looping of the vessels, which press against the bone and cause its atrophy. *Virchow* and others believe that these protuberances correspond to the nutrient territory of certain bone-cells, which in this process aid in reabsorption of the bone. I think I have refuted this, by showing that even dead portions of bone and ivory are also affected by the inflammatory new formation; we shall speak more of this when treating of pseudarthrosis. At present it is not known how the lime-salts are dissolved in this process; I think probably the new formation in the bone develops lactic acid, which changes the carbonate and phosphate of lime into soluble lactate of lime, and that this is taken up and removed by the vessels; but this is only hypothesis. It would also be possible for the organic basis of the bone, the so-called osseous cartilage, to be first dissolved by the inflammatory neoplasia, and then there would be a breaking-down of the chalky substance, whose molecules would be subsequently removed, even if undissolved. Although I have conversed with many chemists and physiologists on this point, none of them have given me a simple explanation of this process, nor could they indicate any mode of experimenting that might aid in solving the question.

In the above diagrams, if we suppose the fractured surface where there is no periosteum, in place of the surface of the bone, you will understand how the new formation (the young callus) grows from it out of the Haversian canals as above described, similar neoplasia from the other fragment meets and unites with it, as in healing of the soft parts. It is evident that the bone through which the inflammatory neoplasia thus grows must become porous, from the reabsorption that takes place on the walls of the canal; if you macerate a bone in this stage, till the young neoplasia decomposes, the dry bone will appear rough, porous, gnawed, while young bone-substance is deposited on it and in its medullary cavity. I must again repeat that in drawings and descriptions we have, for the sake of clearness, made the callous formation appear much more extensive than it really is, and that here, as in wounds of the soft parts, the regenerative processes do not usually extend very far or very deep, but are merely enough for healing, rarely in excess. In this whole explanation we have not mentioned the bone-cells or stellate bone-corpuscles; I am convinced that they have as little to do with these processes as the fixed connective-tissue cells, and that the bone-substance, like the soft parts, is dissolved by a certain amount of inflammation, and replaced by new.

So far we only know the neoplasia in the state where it consists essentially of cells and vessels, as the soft parts do under the same circumstances; if there was retrogression to a connective-tissue cicatrix here as there is there, we should have no solid bone formed, but a connective-tissue union, *pseudarthrosis* (from ψευδής, false; ἄρθρωσις, joint), a false joint; we shall hereafter describe these exceptional cases. Under normal circumstances the neoplasia now ossifies, as you already know. This ossification may either occur directly or after the inflammatory neoplasia has been transformed to cartilage. You know that both of these modes are seen in normal growth of the bone; direct ossification of young cell-formation, for instance, in the periosteum of the growing bone, or formation of cartilage with subsequent ossification, as at first in the entire skeleton and in growth of the bones lengthwise. Callus from fractures varies greatly in this respect in men and animals. In rabbits the callus is always changed to cartilage before ossification, as it also is in children. In old dogs the callus usually ossifies directly, as in the human adult; we are far from knowing the causes of these differences. To obtain a histological representation of these processes, let us return to our former diagram (Fig. 49); now imagine that the cells, lying in the spaces caused by reabsorption in the Haversian canals and surface of the bone, soon ossify and first fill these spaces (Fig. 50), then collect on the surface and in the medulla,

FIG. 50.

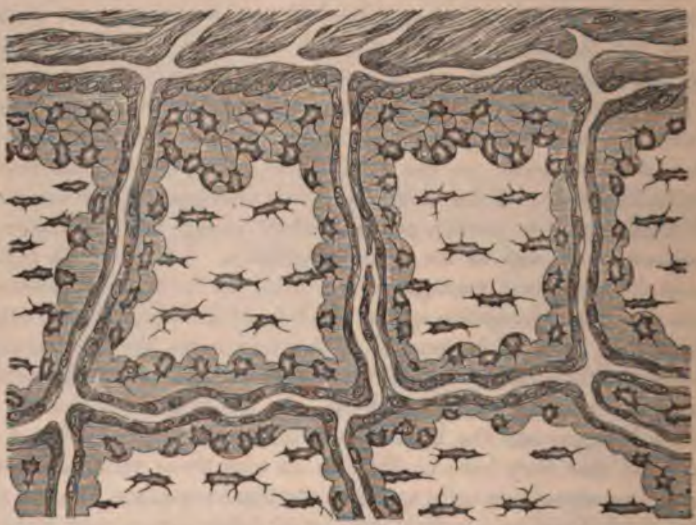
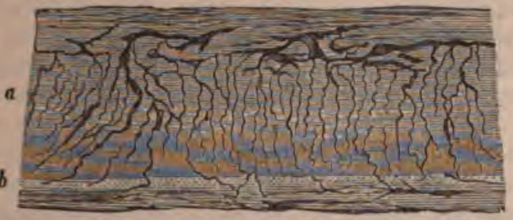


Diagram of ossification of inflammatory neoplasia on the surface of the bone and in the Ha
sian canals. Osteoplastic periostitis and ostitis. Magnified 400 diameters.

and thus form the external and internal callus. Periostitis a ostitis, which lead chiefly or exclusively to the formation of n bone, we call osteoplastic; in the present case the callus is the res of this.

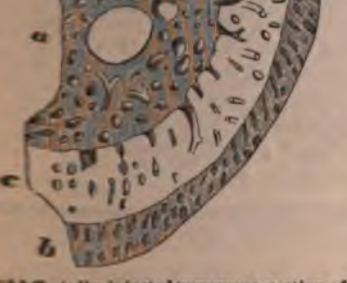
As previously remarked, the periosteum is used up in the neoplasia and in ossifying callus, in its place, externally around the callus, a thick connective-tissue layer develops, from which new periosteum is formed. I will show you a few more preparations in explanation of the process in the periosteum. You see (Fig. 51) the peculiar course of the vessels almost at right angles to the bone, which enter the bone through the young callus. The ossification of the callus begins, mantle-like, around these vessels, and the little columns which first appear in the external callus are thus formed (see remarks on Fig. 44).

FIG. 51.



Artificially-injected external callus, of slight thickness, on the surface of a rabbit's tibia, in the vicinity of a five-day-old fracture. Longitudinal section—*a*, callus; *b*, bone. Magnified 20 diameters.

FIG. 52.



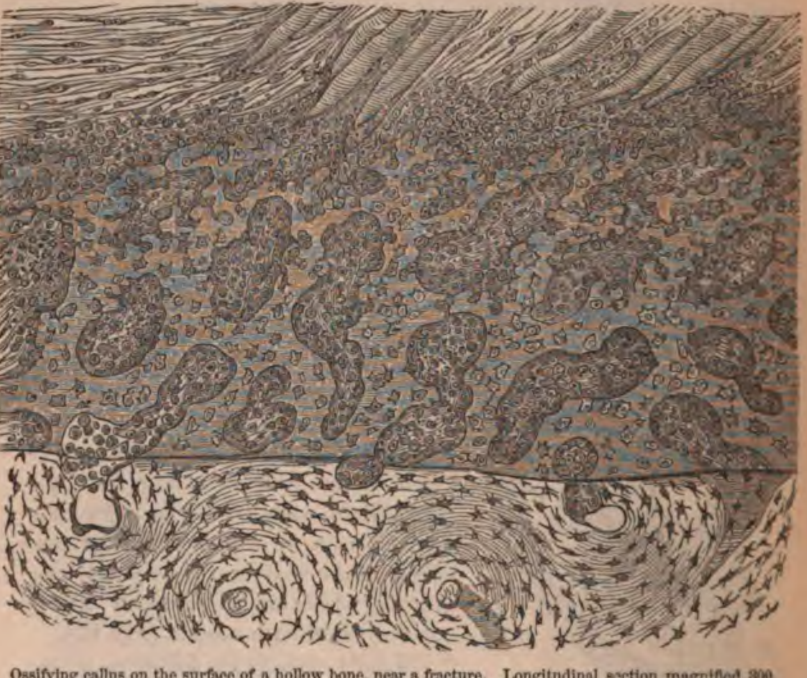
Artificially-injected transverse section of the tibia of a dog, from the immediate vicinity of an eight-day-old fracture. *a*, internal callus; *b*, external; *c c*, cortical layer of the bone. Magnified 20 diameters.

You have a good representation of the formation of external (periosteal) and internal (endosteal) callus in the following (incomplete) transverse section of the tibia of a dog, from the immediate vicinity of an eight-day-old fracture, in which you must also observe the vessels of the cortical substance, which are considerably dilated as compared with normal (Fig. 52).

Lastly, observe the following preparation. It is an eight-day-old, already ossified, external callus on the surface of the tibia of a dog, magnified 250 times (Fig. 53).

If we now view the process as a whole, we see that the cell infiltration in the bone itself, as well as in all the surrounding parts, aids in the formation of callus, and that hence the periosteum plays no exclusive osteoplastic *rôle*. This might have been concluded *a priori*, because if the periosteum alone formed the external callus, as was formerly supposed, the portions of the bone free of periosteum, as those places where tendons are attached to the bone, could form no callus; this is directly contradicted by observation. In normal growth, also, the periosteum does not by any means play the important part ascribed to it in the formation of bone; for we may just as correctly regard the layer of young cells lying on the surface of the bone, and extending into the Haversian canals, as belonging to the bone, as to refer it to the periosteum.

FIG. 53.



Ossifying callus on the surface of a hollow bone, near a fracture. Longitudinal section magnified 300. As appears, the ossifying callus is not limited to the periosteum, but extends in between the muscles.

Recent investigations concerning the growth of bones, made by *J. Wolff*, render it very probable that they increase in all directions by interstitial deposit of young osseous tissue, and hence that growth by apposition through the epiphyseal cartilages and periosteum can no longer be regarded as the sole source of increase in length and thickness; such a mode of growth is placed beyond doubt by *Wegner's* excellent work on the osteoplastic action of phosphorus on growing bones.

I will not conceal from you that the view which I have obstinately maintained, that the bone-cells in new osseous formations do not proliferate, but remain quite passive, is much disputed; since *Cohnheim* has shown the passiveness of the stabile connective-tissue corpuscles in inflammation, there does not seem so much strangeness about my view, which was advanced years ago, and was founded on numerous observations; still, the explanation of the preparations in question is not simple enough to permit only one view. Recently, by very careful investigations about the histological changes during the transformation of provisional into definitive callus, *Lossen* has tried to show that the bone-cells in the former take an active part in the formation of vascular canals in the latter by enlarging and changing position. I can agree with this entirely without abandoning the above views, for the provision-callus is like the young osteophytes of calcified connective tissue, like certain boundary-layers between cartilage and bone. I have no doubt that the cells of this "osteoid cartilage" (*Virchow*), like the cells of hyaline cartilage, proliferate to true bone. But this is not the place to enter more deeply into the histological details, which, great as is their intrinsic interest, have no essential influence on the definitive formation of the new development of bone.

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## LECTURE XV.

Treatment of Simple Fractures.—Reduction.—Time for applying the Dressing, its Choice.—Plaster of Paris and Starch Dressings, Splints, Permanent Extension.—Retaining the Limb in Position.—Indications for removing the Dressings

We shall pass at once to the treatment of simple or subcutaneous fractures, especially fractures of the extremities, for these are by far the more frequent, and they particularly require treatment by dressings, while those of the head or trunk are to be treated less by dressings than by appropriate position, as is taught in the lectures on special surgery and in the surgical clinic.

The indications we have to consider are, simply to remove any dislocations and to keep the fractured extremity in the correct anatomical position till the fracture is healed.

First, the fragments are to be replaced; sometimes this may be unnecessary, as when there is no dislocation, for instance, in some fractures of the ulna, fibula, etc. In other cases it is very difficult, and cannot always be done perfectly. The obstacles to the reposition may be in the position of the fragments themselves; one fragment may be wedged into another, or a small fragment lies between the chief ones, so that the latter cannot be brought together accurately; fractures of the lower articular extremity of the humerus are very obstinate in this respect, for small fragments may be so dislocated that neither flexion nor extension of the elbow-joint can be performed perfectly; hence its functions remain permanently impaired. Muscular contraction forms a second obstacle to the reposition of the fragments; the patient involuntarily contracts the muscles of the broken limb, thus rubs the fragments together or presses them into the soft parts, causing severe pain; this muscular contraction is occasionally almost tetanic, so that, even by great force, it is hardly possible to overcome the opposition. Indeed, formerly these difficulties were, to some extent, insurmountable; and, although attempts were now and then made to attain the object by dividing tendons and muscles, it was often only possible to attain an imperfect reposition. All these difficulties were at once removed by the introduction of chloroform as an anæsthetic. Now, in all cases where we do not readily succeed in reposition, we anæsthetize the patient with chloroform, till his muscles are perfectly relaxed, and we can then usually place the fragments in position without difficulty. Some surgeons go so far as to use chloroform in almost all cases of fracture, partly for the examination, partly for the application of the dressing. This is unnecessary, and may even prove very unpleasant, for some persons, especially those in the habit of drinking, at a certain stage of the anæsthesia are affected with spasmodic contractions of the extremities, so that, in spite of being carefully held by strong assistants, they rub the fractured ends against each other with fearful force, and we must be very careful that a sharp fragment does not pierce the skin. This should not frighten you from using chloroform in fractures, when it is necessary, but simply warn you against being too free with it. The method of reposition is usually as follows: The fractured part is grasped by two strong assistants at the joints above and below the point of fracture, and regular, quiet traction employed, while the surgeon holds the extremity at the point of fracture, and, by gentle pressure, attempts to force the fragments into position. All sudden, impulsive, forced traction is useless, and should be avoided. Here you have to notice two technical expressions; we term the traction on the lower part of the extremity, *extension*, that on the upper part, *counter-extension*. In fractures, these are both made by the hands while in dislocations we must occasionally resort to different mechanical appliances. By the above method accurate reposition will only be impossible when, from excessive swelling or from peculiarly unfavorable dislocation of the fragments, we are unable to correctly recognize the variety of the displacement.

From our present ideas, which are based on a large number of observations, the sooner reposition is made after the occurrence of the fracture, the better; we then at once apply the bandage. This was not always the belief, but formerly the adjustment of the fracture and the application of the dressing were delayed till the disappearance of the swelling, which almost always occurs if a dressing is not at once applied. It was feared that under the pressure of the dressing the extremity might mortify, and the formation of callus would be hindered; with certain cautions in the application of the dressing, the former may very readily be avoided, and there is little truth in the latter belief. Regarding the choice of the dressing also, surgeons have of late reached an almost unanimous opinion. *It may be regarded as a rule, that a solid, firm dressing should be applied as early as possible in all cases of simple subcutaneous fractures of the extremities;* this may be changed altogether two or three times, but in many cases does not need renewal. This mode of dressing is called the *immovable* or *fixed*, in contradistinction to the *movable dressings*, which must be renewed every couple of days, and are only provisional dressings.

There are several varieties of firm dressings, of which the most serviceable are the plaster of Paris, starch, and liquid glass. I shall first describe the plaster dressing, and show its application, as it is the one most frequently used, and answers all requirements in a way that can scarcely be improved.

*Plaster of Paris Bandage.*—After adjustment of the fragments, the broken limb is extended and counter-extended by two assistants, then one or more layers of wadding applied over the point of fracture, and over parts where the skin lies directly over the bone, as over the crest of the tibia, the condyles, and malleoli. Now it is best to envelop the limb with a new fine flannel roller-bandage, so as to make regular pressure on it, and cover all parts that are to be surrounded by the plaster-bandage. In hospital and poor practice, where we cannot always have flannel, we may use soft cotton or gauze bandages. Now comes the application of the plaster-bandages prepared for the purpose; the plaster-bandage that I here have is cut from a very thin gauze-like stuff; it is prepared by sprinkling finely-powdered plaster (modelling plaster) over the unrolled bandage and then rolling it. In private practice a number of these bandages of various sizes may be prepared beforehand and kept in a well-closed tin box. Here in the hospital, where these plaster-bandages are much used, they are prepared two or three times a week. This bandage you place in a basin of cold water and let it soak through, then apply it like any roller-bandage to the extremity prepared as above described. Three or at most four thicknesses of this plaster-bandage suffice to give the dressing the requisite firmness. In about ten minutes good plaster becomes stiff enough for us to lay the extremity loose on the bed; in half an hour or an hour, the dressing becomes as hard as stone and quite dry; the time required for hardening depends partly on the quality of the plaster, partly on how much you have moistened the bandage. After many comparisons with other modes of applying the plaster-bandage, I have found this the most practical; but I must mention some modifications of the way of handling the plaster and of the material of the bandage. For instance, we may rub the plaster into the common muslin or flannel bandages, which makes the dressing somewhat heavier and firmer; but this is not necessary and the loose gauze is very much cheaper than muslin-bandage. If the bandage does not appear sufficiently firm, we may apply a layer of plaster-paste over the dressing; this plaster-paste is to be made with water, and spread on the bandage very quickly with the hand or a spoon; it should not be prepared till we wish to use it, as it stiffens very quickly. The plaster-dressing as made with roller-bandages was first introduced by a Dutch surgeon, *Mathysen;* this method was first published in 1832; but it has only become well known since 1850; it has been spread through Germany chiefly by the Berlin school. A different mode of applying the plaster-dressing is by different strips of bandage; *Pirogoff* first hit on this method from lack of bandages in the army; all kinds of material were cut into the shape of splints, then drawn through thin plaster-paste and laid on the broken limb, then the whole was coated with plaster-paste and a firm capsule was thus made. Subsequently the same surgeon made a special method of this; he cut old coarse sail-cloth into certain patterns for each limb, and applied it in the above manner. Lastly, the so-called many-tailed bandage of *Scultetus* was used in the same way as a plaster-bandage. The foundation of the bandage has also been modified in various ways; it has even been used without wadding or any under-bandage, the whole limb being simply covered with oil so that the plaster-bandage, being applied directly, might not adhere to the skin by the fine hairs. Others have employed thick layers of wadding without any under-bandage. Lastly, thin wooden splints or strips of tin have been lately used in it, as we shall hereafter see; this may have certain advantages in fenestrated bandages.

I have intentionally represented all these modifications of the plaster-bandage as only exceptionally useful, all of them having certain objections as compared with the method first described. A more careful criticism of these modifications here would lead us too far.

For persons unskilled in the matter, the removal of the plaster bandage is quite difficult, but you may see that any of my nurses will do it with astonishing quickness. It is simply done as follows: with a sharp, strong garden-knife we divide the plaster-bandage, not perpendicularly but rather obliquely, as far as the under-bandage, then remove the bandage entire, like a shell; we may also employ the plaster-scissors proposed by *Szymanowski* or those of *Bruns*. We use this capsule in some other cases as a provisional dressing.

*Starch-Bandages.*—Before plaster-bandages were known, we had in the starch-bandage an excellent material for the immovable dressing. The starch-bandage was perfected and introduced chiefly by the Belgian surgeon *Seutin* († 1862); it is only during the last twelve years that it has given place to the plaster-dressing, but it is still used occasionally. The application of the wadding and under-bandage is the same as in the plaster-dressing, but then we apply splints, cut from moderately thick pasteboard and softened in water, to the limb, and fasten them on with bandages thoroughly soaked in starch-paste; we now apply wooden splints till the dressing has hardened, which at the ordinary temperature requires about twenty-four hours. Compared to the plaster-dressing this has the disadvantage of hardening much more slowly; we may improve this somewhat if we use *gutta-percha splints* instead of pasteboard, these may be softened in hot water, and adapted to the extremity. Gutta-percha bands, such as are used in factories, are very useful as splints. It cannot be denied that the introduction of gutta-percha into surgery is to be regarded as a great advantage; but it is too costly to be used in practice for every simple fracture, although thick splints of this material harden even quicker than plaster. The dressing with roller-bandages prepared with plaster is so cheap and firm that it will certainly not be displaced again by starch-bandages, now that it has been introduced into practice.

Instead of plaster, solutions of dextrine, pure white of egg, or simple mixture of flour and water, were formerly employed; they have all gone out of use, but it is well for you to know the usefulness of these substances, which are in every house, and which we may well employ as provisional dressings.

*Liquid-glass Dressings.*—Instead of starch, we may employ the liquid glass of the shops (silicate of potash). On applying the d ing, we paint this on the muslin-bandages with a large bru having made a substratum of wadding as above described. T glass dries quicker than starch, but not so soon as plaster, nor does it become as hard as the latter; this dressing does for fractures with no tendency to displacement; if we wish to fix dislocated fragments of bone by the liquid-glass dressing, we must strengthen it by applying splints.

I doubt not the time will soon come when every country physician will always keep a few plaster-splints ready prepared; in spite of them, *provisional dressings* remain useful. These consist of bandages, compresses, and splints, of various materials. You may make splints of thin boards, shingles, cigar-boxes, pasteboard, tin, leather, firmly-plaited straw, the bark of trees, etc., and, for bandages, must often content yourselves with old rags, muslin, torn into strips and sewed together; hence, in the practical courses on bandaging, it is necessary for you to learn to make use of the most varied materials.

It is not our intention here to introduce to you every thing that may be used in the way of dressing, but I must still speak briefly of a few things. As may be readily seen, the object of the splints is to make the bone immovable by supporting it firmly on various sides; this may be attained by external, internal, anterior, and posterior, narrow wooden splints; we may, however, employ hollow splints, so-called gutters. Hollow splints are only good when made of pliable material, as leather, thin sheet-iron, wire-gauze, etc.; an absolutely stiff, hollow splint would only do for certain persons. Besides these mechanical aids, there is another method of keeping broken limbs in position, namely, *permanent extension*. This is particularly indicated in cases where there is great tendency to shortening, to *dislocatio ad longitudinem*. Attempts have been made to attain this extension by attaching weights by various mechanical contrivances, by continued traction made by weights hung to the injured limb, by the double-inclined plane, where the weight of the leg is used as the extending weight. Since, during the past two years, I have unexpectedly seen such excellent effect from permanent extension with weights in painful contractions at the hip and knee joints, I am compelled to believe that this method may also eventually prove very serviceable for the gradual adjustment of dislocated fragments of bone. Among the arrangements of this nature with which I am acquainted, *V. Dumreicher's* so-called railroad apparatus best fulfils the object of permanent extension, but it is too costly and complicated to come into extensive use in private practice; it is, doubtless, the intention of the inventor to employ it chiefly in cases where the dislocation is difficult to overcome. [Dr. *Gurdon Buck's* apparatus for fractured thigh is about as efficacious and much simpler.] The double-inclined plane, represented by a thick roller-cushion applied under the hollow of the knee, may occasionally be employed as a suitable fixation apparatus in fracture of the neck of the femur in old persons.

We must still mention some auxiliary appliances which we have to employ to keep the broken limb in good position after it has been dressed; for the upper extremity, in most cases, a simple, properly-applied cloth, a *mitella*, or *sling*, in which the arm is laid, suffices. Patients with fractured arm or forearm may be permitted to go about with a plaster-bandage and a sling during the entire treatment, without interfering with the favorable healing.

For keeping broken lower extremities in position, there are a number of mechanical aids, of which the following are the most serviceable: *sand-bags*, narrow sacks filled with sand, about the length of the leg; these are placed both sides of the firm dressing, so that the limb may not move from side to side; for the same purpose we may use long, three-sided pieces of wood, cut prismatically, which are laid together, so as to form a gutter. For some cases a sack, loosely filled with chaff or oats, is sufficient; we make a hollow in it lengthwise, and the leg is to be placed in this. If we desire firmer supports, we use *fracture-boxes*, narrow, long, wooden boxes, open at the upper end, so that the leg may be placed in them; and the sides are made to turn down, so that the extremity may be carefully inspected, without moving it; the elevation of these fracture-boxes may be suited to the convenience of the patient. Lastly, we must mention the *swing*, which is usually made with a gallows, or strong bow, that is brought over the foot of the bed, and to which the limb is suspended in any sort of a fracturé-box, or hollow splint [or Dr. *Nathan Smith's* anterior splint], so that it may swing about; in restless patients especially, this has certain advantages. All these apparatuses, which, although more rarely employed than formerly, are still occasionally useful, you must learn to apply; you will have opportunity for this in the surgical clinic. Of late we rarely apply these apparatuses in the lower extremity, as my former assistant, Dr. *Ris*, who has brought the application and elegance of the plaster-bandage to an extraordinary state of perfection, applies a well-padded wooden splint, three or four inches wide, to the under side of the leg, making it reach somewhat below the heel and as high as the knee, or, in fractures of the thigh, as high as the middle of the thigh. The limb lies firmly on this board, if the mattress be not too uneven; if we wish to attain still greater firmness, we may lay a board the width of the bed over the lower third of the mattress, and on this place the limb, with its plaster-dressing and supporting splint. In the numerous double fractures of both lower extremities that came to the Zürich hospital, this supporting apparatus did excellent service.

The old form of plaster-moulds has been recently strongly advocated again by Dr. *M. Müller;* we have tried it again, but it bears no comparison with the plaster-bandage.

*Seutin* tried to increase the advantages of firm dressings by giving aids that might enable patients with fractured lower limbs to go about to some extent. For instance, a patient with a broken leg may have a broad leather strap passing over the shoulder, and buckled just above the knee, so that the foot will not touch the floor, and then let him go on crutches. But I advise you not to carry these experiments with your patients too far; at all events, I only allow my patients to make such attempts three weeks after the occurrence of the fracture, otherwise œdema readily occurs in the broken limb, and some patients are so clumsy in the use of crutches, that they are apt to fall, and, although this may only cause slight concussion of the limb, it is still injurious.

Lastly, we have to discuss how long the dressing should be left on, and the causes that might induce us to remove it before the cure is complete. The decision as to whether a dressing is too tightly applied is entirely a matter of experience; the following symptoms must guide the surgeon: If there be swelling of the lower part of the limb, as of the toes or fingers, which are usually left exposed, if these parts become bluish red, cold, or even senseless, the dressing should be removed *at once.* If the patient complains of severe pain under the dressing, it is well to remove it, even if we can see nothing to cause it. In judging of the exhibitions of pain, we should know the patients; some always complain, others are very indolent, and show their feelings but little; however, it is better to reapply the bandage several times uselessly than once to neglect its removal at the right time. I cannot too strongly urge you always to visit, within twenty-four hours at most, every patient to whom you apply a fixed dressing; then your patient will certainly not come to grief, as unfortunately too often happens, from the carelessness and laziness of his surgeon. A series of cases has been published where, after the application of a firm dressing, the affected limb mortified, and required amputation; from these cases it was decided that firm dressings were always improper, while the fault was chiefly due to the surgeon. Just think how little trouble we have in treating fractures now, compared to former times, when the splints had to be renewed every three or four days; now you need only apply a dressing once But you must not think you have got rid of all trouble in the application of dressings. The application of the firm dressing requires just as much practice, dexterity, and care, as did dressing with splints. If you are first called to a fracture when it is two or three days old, when there is already considerable inflammatory swelling, you may even then apply the firm dressing, but must apply it more loosely, and with plenty of wadding. This dressing will be too loose, and should be renewed in ten or twelve days, when the swelling has left the soft parts. It will chiefly depend on the looseness of the bandage, and the greater or less tendency to dislocation, when and how often the dressing should be removed during the treatment. Swelling, if not accompanied by considerable contusion, is no contra-indication to a carefully-applied firm bandage; nor do large or small vesicles, full of clear or slightly-bloody serum, present any great objection; such vesicles result not unfrequently from contused fractures with extensive rupture of the deep veins, since, from obstruction to the flow of venous blood, the serum readily escapes from the capillaries, and elevates the hard layer of the epidermis into a vesicle; we puncture these vesicles with a needle, gently press out the fluid, and apply some wadding, and they soon dry up. It is the same with slight superficial excoriations of the skin; we are only rarely obliged to remove the dressing and apply another, when new vesicles form, as we may know by the pain.

The length of time that a firm dressing must remain on for the different fractures you will learn partly in the clinic, partly from special surgery; I simply mention here, as the limits, that a finger may require a fortnight, a thigh sixty days, or more, for healing. If you apply the plaster-dressing immediately after the fracture, dislocation having been completely removed, the provisional callus will always be less, and hence firmness result later, than where there is some dislocation and the dressing is applied later; but this has no effect on the formation of definitive callus, and the actual union of the fractured ends of the bone.

[The efficacy of plaster bandages depends greatly on the quality of the material; we may easily procure good modeling plaster, but it spoils if kept some time, without being entirely protected against air and moisture. Now in *tripolith* we have a material possessing the good qualities of plaster, without its disadvantages; this is a grayish-black, fine powder of secret composition, to be used just as plaster is, but having the following advantages: 1. It may be kept any length of time, even in an open vessel; 2. It is lighter than plaster, hardens even more rapidly, and, once hardened, resists moisture; and, 3. Is cheaper than plaster. It is used in just the same way as plaster, which it will probably supplant.

When using starch bandages we may employ organtine, or lining gauze, which is coarse-meshed and starchy, consequently stiff; when soaked in water it becomes soft, then quickly dries and hardens;

several layers are to be applied, and they unite together into a firm but light capsule. This simplifies the application of starch bandages, and they look better.

A step between these fixed and movable dressings is the gutta-percha splint; for this we use sheet gutta-percha, cut into splints of proper size; dip these in very hot water till they become soft and flexible, then apply to the limb after smearing it with oil, and over this a moist bandage. As the gutta-percha cools it hardens. Sometimes it is well to apply a firm splint till the other is perfectly hard. The advantages of this splint are its accurate adjustment and impermeability to water; the objection is its cost, although the same splint may be used several times.

In some parts of the body extension and counter-extension by fixed dressings or splints are very difficult, and permanent extension by *Volkmann's* method is resorted to. Two long pieces of adhesive plaster two inches wide are applied along the sides of the limb and kept in place by circular turns, a roller bandage being put over the whole; a piece of wood is placed below the foot like a stirrup, and to this a bag of sand or shot is attached by a cord, which passes through a vertical pulley fastened to the foot of the bed. The limb is placed in a tin trough, running by means of small rollers on a wooden frame. [This is a slightly complicated *Buck's* apparatus.] The weight may vary from four to ten pounds, according to the strength of the patient and the amount of dislocation. This weight is divided up over the surface of the limb, and is well borne. Permanent extension may be resorted to for fractures of the humerus, but is not often used in such cases, unless the patient is kept in bed by other injuries.

If there be considerable swelling, with crushing and suffusion of the skin, and formation of vesicles full of bloody serum, before applying a fixed dressing certain precautions are necessary. Circumscribed gangrene of the skin may result, changing the subcutaneous into an open fracture; to prevent infection and decomposition in case this occurs, after cleansing and disinfecting the skin, the crushed part should be covered with a thick layer of *Lister's* gauze and padding, then the plaster bandage applied. Under this treatment the gangrenous skin dries to a crust, which separates with very little suppuration. If pain, swelling, and fever set in, the dressing should be at once removed.

We may here discuss an important question: Are patients feverish after a subcutaneous fracture; and, if so, of what prognostic value is the fever? It is only a short time since observations have been made on this point. When the patient has had his fracture dressed, he usually feels comfortable ; so it was taken for granted that he had no fever. But numerous measurements have shown that during the first days after the injury the evening temperature is often 102° ; though this is almost exclusively in cases of fracture of the long bones, and when there is great extravasation of blood ; but even where the symptoms of this complication fail, we must suppose that there is a certain amount of extravasation from the ruptured vessels of the medulla, and this must be reabsorbed. In injuries of the soft parts we have seen that reabsorption of the primary secretions induced fever, hence we need not wonder at finding elevation of temperature after simple fractures ; but, as these primary secretions are not very irritant, patients are not particularly uncomfortable, and the temperature subsides after four or five days ; if it recurs in the second or third week, with pain, swelling, and redness, something else must be taking place, and the dressings should be removed.]

# CHAPTER VI.

## *OPEN FRACTURES AND SUPPURATION OF BONE.*

Difference between Subcutaneous and Open Fractures in regard to Prognosis.—Varieties of Cases.—Indications for Primary Amputation.—Secondary Amputation.—Course of the Cure.—Suppuration of Bone.—Necrosis of the Ends of Fragments.

We shall now pass to complicated or open fractures.

When we speak simply of *complicated fractures*, we usually mean only those accompanied by wounds of the skin. Strictly speaking, this is not exact, because there are other complications, some of them much more important than wounds of the skin. If the skull be fractured, and part of the brain-substance crushed, or some ribs broken and the lung wounded, these are also complicated fractures, even though the skin should remain uninjured. But, since in these cases the complications themselves are more important for the organism than the fracture is, we usually term such cases contusion of the brain, or injury of the lung, with fracture of the skull or ribs. But we shall not here enter on the subject of injuries of internal organs by fragments of bone, because very complicated states of disease are occasionally induced in this way, whose analysis you would not now understand. For the present let us limit ourselves to fractures of the extremities, accompanied by wounds of the skin, which we shall call open fractures, and which will give us trouble enough in their course and treatment.

In speaking of the course of simple contusions without wounds, and of contused wounds, I have already shown you how readily reabsorption of extravasated blood and the healing of contused parts g on, as long as the process is subcutaneous, but how much the conditions change if the skin also be destroyed. The chief dangers in suc cases are, as you may remember, decomposition in the wound, exte sive necrosis of crushed or dead parts, progressive suppuration, an accompanying protracted, exhausting fever, while we have scarcel mentioned the severe general diseases, erysipelas, putrid-blood poisoning, pyæmia, tetanus, and delirium tremens. The difference between contusions and contused wounds is even more strongly marked in simple and compound fractures, as regards course and prognosis. While in many cases we can scarcely call a person with simple fracture sick (we have not spoken of fever there, for it is rarely high), and under the present mode of treatment such an injury is rather an inconvenience than a misfortune, a compound fracture of a large bone of an extremity, or sometimes even of a finger, may induce severe, and too frequently fatal, disease. But, not to alarm you too much, I will at once add that there are many grades of danger even in open fractures, and, moreover, that their treatment has been much improved of late.

It is very desirable and important, but not always possible, to make a correct prognosis about an open fracture at once. The life or death of the patient may occasionally hang on the choice of the treatment the first few days, so that we must study this subject more accurately. The symptoms of an open fracture are of course essentially the same as of the subcutaneous, except that discoloration from extravasated blood is often wanting, because at least part of the blood escapes through the wound. The fractured ends not infrequently project from the wound, or lie exposed in it, so that a glance may suffice for the diagnosis of an open fracture. But this is not enough. We must do our best to ascertain how the fracture was caused, whether by direct or indirect force, and how great the force; if it was accompanied by crushing and twisting; whether arteries and nerves have been torn; if the patient lost much blood, and what is his condition at present. There are cases where we can say, at the first glance, healing is impossible; amputation must be resorted to. When a locomotive has run over the knee of an unfortunate railroad hand, when a hand or forearm has been caught in the wheels or rollers of machinery, when a premature explosion in blasting stone has crushed or torn off a limb, or hundred-weights have completely mashed a foot or leg, it is not difficult for the surgeon to decide at once on primary amputation, and usually in such cases the state of the limb is such that the patients also, though with a sad heart, quickly consent to the operation. These are not the difficult cases. And in other cases it may be just as easy to foretell, with considerable certainty, the probability of a favorable cure. For instance, if fracture of the leg from indirect force has followed too great bending of the bone, the broken pointed end of the crest of the tibia may puncture and force through the skin; in such a case there is no contusion, but simply a tear through the skin. When a pointed body strikes forcibly against a small portion of a limb, and injures bone and skin, the whole extremity may be greatly shaken; but the extent of the injury is not great, and most of such cases terminate favorably under suitable treatment. The questionable cases lie between these two extremes. In cases where there is some contusion, but only a slight amount evident, and the skin is only injured at a small spot, it will be very difficult to decide whether healing should be attempted or amputation be resorted to, and the peculiarity of the individual case alone can settle the question. Of late the tendency is increasing rather to try to preserve the limb in these doubtful cases than to amputate one that might possibly have been saved. This principle is certainly justified on humane grounds; but it cannot be denied that this conservative surgery may be practised at the cost of life, and that we cannot with impunity vary too much from the principles of the older surgeons, who generally preferred amputation in these doubtful cases. Besides mode of origin of the injury, and the amount of accompanying contusion, the importance in any given case depends on whether we have to deal with deep wounds, with fractured bones lying far down among the muscles, or with bones lying near the skin, as the danger of suppuration depends greatly on the depth and extent of the bone-injury. Thus, an open fracture at the anterior part of the leg is of more favorable prognosis than a similar injury of the arm or forearm. Open fractures of the thigh are the most unfavorable; indeed, some surgeons always amputate for such injuries. Large nerve-trunks are rarely torn in fractures, and, when they are, it does not seem to have much effect on the cure; and experiments on animals, as well as observations on man, show that bones may unite normally in paralyzed limbs. Injury of large venous trunks, as of the femoral vein, causes hæmorrhage, which may be readily checked by a compressing-bandage, it is true, but may prove dangerous when the blood effused between the muscles and under the skin begins to decompose. Rupture of the arterial trunk of a limb occasionally leads at once to considerable arterial hæmorrhages; but this is not a necessary sequence; for, as previously shown, a thrombus quickly forms in the crushed artery, so that we do not always have extensive hæmorrhage. But, if, from the nature of the hæmorrhage, we recognize the rupture of an artery, according to principles already laid down, we should either attempt to ligate the artery at the wound, or else at the point of election. Rupture of the femoral artery with fracture of the femur is found by experience to be followed by gangrene, and is an imperative indication for amputation; in a corresponding injury of the arm, recovery may result or gangrene may follow. In fractures of the forearm or leg, even if one or both arteries be ruptured, recovery may take place. Lastly, in the question as to whether we shall try for union, or proceed to amputation, we must consider how useful the limb can be if union results and all unfavorable chances have been overcome. In complicated fractures of the foot and lower part of the leg this question may be particularly important, and it has frequently been necessary to amputate a foot because of the change of form and position resulting after union of an open, comminuted fracture, which rendered it useless for walking. The same thing is to be considered when, in a case of moderately extensive gangrene of the foot, we wish to decide if it should be amputated or not. The dead portion of the foot may be detached in such an inconvenient shape that the remaining stump is neither useful for walking nor for the adaptation of an artificial limb. In such cases we should amputate, for all our methods of amputating are designed for the future application of artificial limbs.

Since the nature of the subject has led us directly to the indications for amputation in injuries, I shall at once proceed to the subject of *secondary amputations.* In the question as to whether a complicated fracture should be amputated or not, you might readily satisfy yourself with the idea that it might be done at any future time if the fears of an unfavorable course should be realized. On this point attentive observation shows that there are two periods for this secondary amputation. The first danger threatens the patient from an acute decomposition about the wound and the consequent putrid intoxication of the blood. The question as to this danger is settled during the first four days; if it arises, and you then amputate (this must be done far above the point of putrefaction), it is just at the most unfavorable period for the operation, for you will *very rarely* succeed in saving your patient. Somewhat more favorable, but still unfavorable as compared with primary amputations (those made within the first forty-eight hours), are the results of amputations made from the eighth to the fourteenth day; they are particularly unfavorable if the symptoms of acute purulent infection, pyæmia, are distinctly present. If the patient has survived two or three weeks, and profuse exhausting suppuration or other local indication for amputation arise, the results are again relatively favorable. When some surgeons have asserted that secondary amputations give better results than primary, they have almost exclusively considered these later secondary amputations. But, if we bear in mind how many patients with open fractures die during the first three weeks, that is, how few of them live till the favorable time for secondary amputations, it seems to me we can have no doubt about the decided advantages of primary amputations. Up to the present time I have rarely found indications for late secondary amputations.

An open fracture may unite in various ways. The skin-wound, as well as the deeper parts, occasionally heals by first intention; this is the most favorable case. Under modern treatment this occurs more frequently than formerly, although, from the nature of the case, the requirements for this result are not often present. Far more frequently (and this is also favorable) the wound only suppurates superficially, and not between and around the ends of the bone, but union of the bone takes place as in simple subcutaneous fracture. The cases where the wound only affects the skin, and does not communicate with the fracture, should not be counted among complicated fractures; but the limits are difficult to trace.

The process of cure must of course differ greatly from the above, if the skin-wound be large, the soft parts greatly contused, so that fragments are detached from them; if the suppuration extends deep between the muscles and around the bone, and even into its medullary cavity; if the fragments are bathed in pus; if half-loose pieces of bone lie about, and longitudinal fissures extend into the bone. The activity of the soft parts will remain essentially the same as in subcutaneous fractures, except that in this case the inflammatory new formation does not directly become callus, but, after detachment of the crushed, necrosed shreds of tissue, granulations and pus are formed, the former of which are transformed to ossifying callus. The form of the callus will not be much changed, except that, where the open suppurating wound exists for a long time, there will be a gap in the callus-ring till it is closed by the after-growth of deep ossifying granulations. Hence the process will terminate far more slowly than in subcutaneous fracture, just as healing by suppuration takes longer than healing by first intention.

Now, what becomes of the ends of the fragments which, partly or entirely denuded of periosteum, lie in the wound? What becomes of pieces detached from the bone, and only loosely attached to the soft parts? As in the soft parts, so here one of two things may happen, according as the ends of the bone are living or dead. In the first and most frequent case, granulations grow directly from the surface of the bone. In the latter, as in the soft parts, plastic activity in the bone occurs on the borders of the living; interstitial granulations and pus form; the bone melts away; the dead end of the bone, the *sequestrum*, falls off. The extent to which this process of detachment goes naturally depends on the extent to which the bone is dead, or, expressed more physiologically, on the extent to which the circulation has ceased from stoppage of the vessels. This extent may vary greatly: it may possibly extend only to the superficial layer of the injured bone: and, since the whole process is called *necrosis*, this superficial detachment of a plate of bone is termed *necrosis superficialis*, while that of the whole fractured end of the bone may be called *necrosis totalis;* but the latter term is more usual for indicating that the entire diaphysis of a long bone, or at least the greater part of it, is detached, and the opposite of this is *necrosis partialis.* The opposite of the above-mentioned necrosis superficialis, which is also termed *exfoliation*, is properly *necrosis centralis*, that is, detachment of an inner portion of bone. Necrosis superficialis and necrosis of the broken ends and partly-detached fragments of the bone are so often combined with suppurating fractures, of which we have to treat here, that we must treat of them in this place. It will at first seem strange to you that vascular granulations should spring from the hard, smooth cortical substance of a long bone. From what has already been said, it will seem possible that, under the influence of this plastic process, the hard osseous tissue should be so dissolved that there may be a spontaneous solution of continuity between the dead and healthy bone. We shall now study more exactly these processes of formation of granulations and of suppuration in bone.

You will remember, from the full description of traumatic suppuration of the soft parts, that in traumatic inflammation the process chiefly depends on free suppuration and extensive formation of new vessels, as well as on direct cell-infiltration from the blood, while the intercellular substance assumes a gelatinous or fluid consistence. Both of these processes can only take place to a slight extent in bone, especially in the firm cortical substance of a long bone, because the firm osseous substance prevents much dilatation of the capillaries which are enclosed in the Haversian canals. I may at once call your attention to the fact that, from this slight distensibility of the vessels in the osseous canals, portions of bone may more readily die than would be the case with the soft parts, because, in case of coagulation of blood, even in the smaller vessels, the nutrition can be only imperfectly kept up by collateral circulation. Moreover, the connective tissue and the vessels in the Haversian canals may be entirely destroyed by suppuration, so that necrosis at the ends of the fragments will be inevitable. Should a vascular granulation-tissue develop on the surface of the bone or in its compact substance, this can only occur as previously described, after the osseous substance (lime-salts as well as organic matter) has disappeared at the point where the new tissue is to appear; hence there must be solution and atrophy of the bone-tissue, just as there are of the soft parts under similar circumstances (see Fig. 39). The whole difference appears chiefly in the difference of time, for the development of granulations on and in the bone takes much longer than in the soft parts. I have already stated that the same process requires much longer in the tendons and fasciæ, which have few vessels, than in the connective tissue, muscles, and skin; in the bone it requires even more time than in the tendons. The constitutional power of the individual, and the consequent so-called vitality of the tissues, are also to be taken into consideration.

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## LECTURE XVI.

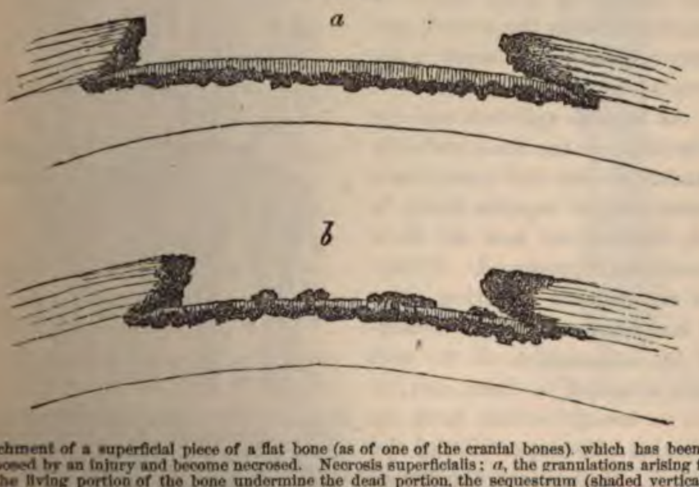
Development of Osseous Granulations.—Histology.—Detachment of the Sequestrum.—Histology.—Osseous New Formation around the Detached Sequestrum.—Callus in Suppurating Fractures.—Suppurative Periostitis and Osteomyelitis.—General Condition.—Fever.—Treatment; Fenestrated, Closed, Split Dressings.—Antiphlogistic Remedies.—Immersion.—Rules about Bone-splinters.—After-Treatment.

When a denuded portion of bone begins to throw out granulations on its surface (which in complicated fractures we can only see when the ends of the fragments are exposed by a large skin-wound, on the interior surface of the leg, for instance), we recognize this with the naked eye by the following changes: For the first eight or ten days after being denuded of periosteum, the bone mostly preserves its pure yellowish color, which, even during the last day of the above period, changes toward bright rose-color. If we then examine the surface of the bone with a lens, we may notice numbers of very fine red points and striæ, which a few days later become visible to the naked eye also; these rapidly increase in size, grow in length and breadth, till they unite and then present a perfect granulating surface which passes immediately into the granulations of the surrounding soft parts, and subsequently participates in the cicatrization, so that such a cicatrix adheres firmly to the bone.

If we follow this process in its finer histological details, which must be chiefly done experimentally, by aid of injected bones deprived of their lime, we have the following result: If the circulation in the bone is maintained near to the surface, there is a rich infiltration of cells into the connective tissue accompanying the vessels in the Haversian canals; this tissue grows, with the vascular loops developing toward the surface, out of the bone at the points where the Haversian canals open externally. The development of this young granulation-mass laterally results at the expense of reabsorbed bone. If we macerate one of these bones with superficial granulations, its surface will appear gnawed and rough; in the living bone, granulation tissue fills the numerous small holes, which all communicate with the Haversian canals. The surface of the bone does not, however, rem in this state, but, while the osseous granulations on the surface condense to connective tissue and cicatrize, in the deeper parts they ossify quite rapidly, so that at the termination of the process of healing the surface of the injured bone does not show a deficiency, but appears denser from deposit of new bone. You see that here too the circumstances are exactly the same as in subcutaneous development of the inflammatory neoplasia. If you look at Fig. 49, and suppose the periosteum removed from the surface of the bone, the new formation (in this case as granulations) will grow fungous-like out of the Haversian canals.

You will understand this better if we now follow more carefully the process of detachment of necrosed portions of bone. Let us return to what we see with the naked eye, and let us suppose we have before us a portion of the parietal bone denuded of soft parts; then, if no granulations, as above described, grow from the bone, we shall have the following symptoms: While the surrounding soft parts and the portion of bone still covered with periosteum have already produced numerous granulations and secrete pus, the dead portion of bone remains pure white or becomes gray or even blackish. It remains some weeks, sometimes two months or more; most proliferant granulations grow around it; cicatrization has already begun in the periphery of the wound, and we cannot decide how the case will terminate, for in the sixth week the surface of the bone may look just as it did the day after injury. Some day we feel the bone and find it

FIG. 54.



Detachment of a superficial piece of a flat bone (as of one of the cranial bones), which has been exposed by an injury and become necrosed. Necrosis superficialis: *a*, the granulations arising from the living portion of the bone undermine the dead portion, the sequestrum (shaded vertically); *b*, the lower side of the sequestrum has been considerably eaten away by the granulations, which have perforated it at various points. Diagram, natural size.

movable; after a few attempts one blade of the forceps may be introduced under it and we lift off a thin plate of bone, under which we find luxuriant granulations; the under surface of this plate is very rough, as if eaten away. Now healing goes on rapidly. It is often long before the cicatrix becomes permanent and solid enough to resist all injuries, such as pressure and friction, but healing often terminates favorably. This is the process that we term *necrosis superficialis* or exfoliation of bone. We are already acquainted with this process in the soft parts; during the first week large shreds of tissue fall from the contused wound, since on the border of the healthy tissue there is an interstitial development of granulation, by which the tissue is detached; the process is the same here. In a bone deprived of its lime we may readily examine these processes anatomically. The inflammatory neoplasia, or granulation tissue, develops on the margin of the healthy bone in the Haversian canals. The accompanying figure (Fig. 55) may represent to you the details of this process.

If you have fully understood what has been said, it only requires a slight stretch of imagination to see how the same process of detachment of a fragment may extend through the entire thickness of bone; that is, how (and here we come back to complicated fractures) a variable length of the fractured end of a bone may be entirely detached, when it is incapable of living. When the bone in question is thick, this process requires several months; but at last we may find even large pieces of bone movable in the wound, and remove them as we would a superficial bony plate.

As regards splinters entirely detached from the bone, and only attached to the soft parts, their future fate, as regards living or not, depends on how far their circulation is preserved. If they are not capable of living, they at last become entirely detached by suppuration of the soft parts attached to them, and often, as foreign bodies, keep up irritation and suppuration of the wound. If they are capable of living, they produce granula-

Fig. 55.

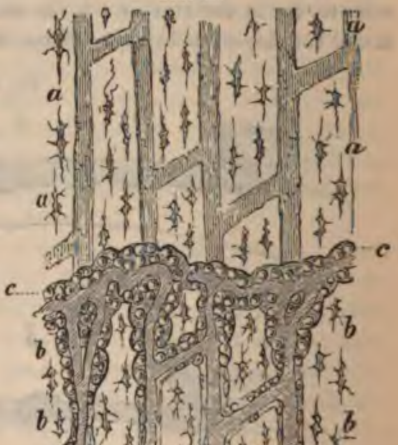


Diagram of detachment of a necrosed portion of bone. Magnified 300. *a*, necrosed portion of bone; *b*, living bone; *c*, new formation in the Haversian canals, by which the bone is detached. Compare Fig. 36.

tions on the free surface; these subsequently ossify and unite with the other callus, forming around the fractured ends.

To represent the relation of the formation of callus to this process of detachment of the necrosed ends of the fractured bone, I present the following figure (Fig. 56).

The fragments of the broken bone are not accurately adjusted, but displaced somewhat laterally; the ends of the fragments have both become necrosed, and nearly detached by interstitial proliferation of granulations on the borders of the living bone. The whole wound is lined with granulations, which secrete pus that escapes at *d*. In both fragments, an inner callus (*b b*) has formed, which, however, from suppuration of the fractured surfaces, has not yet been soldered together. The outer callus (*c c*) is irregular, and interrupted at *d*, because the pus escapes here from the first. When the granulations grow so strongly as to fill the entire cavity, and subsequently ossify, healing is completed, and the final result is just the same as in the healing of subcutaneous fractures. For this to take place the necrosed portions of bone must be removed, for experience shows they cannot heal up in the osseous cicatrix. This elimination of the sequestrated

FIG. 56.

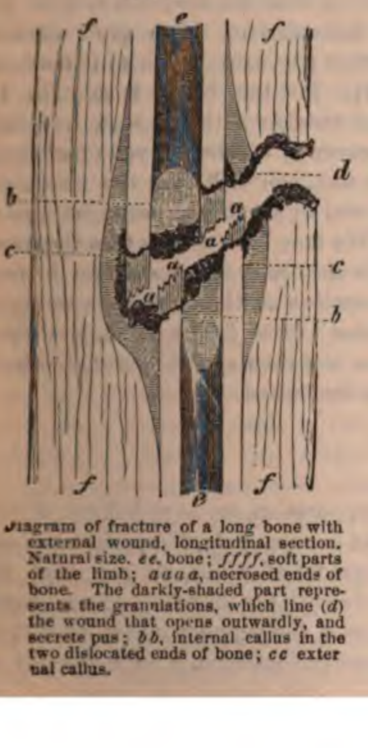
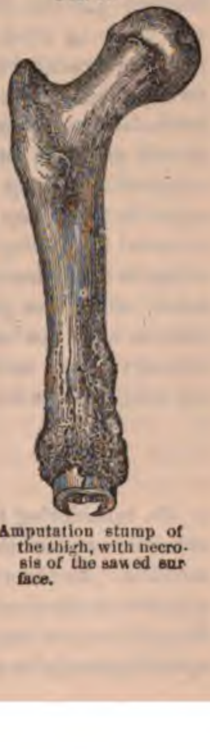


Diagram of fracture of a long bone with external wound, longitudinal section. Natural size. *ee*, bone; *ffff*, soft parts of the limb; *aaaa*, necrosed ends of bone. The darkly-shaded part represents the granulations, which line (*d*) the wound that opens outwardly, and secrete pus; *bb*, internal callus in the two dislocated ends of bone; *cc* external callus.

FIG. 57.



Amputation stump of the thigh, with necrosis of the sawed surface.

fragments takes place either by reabsorption or by artificial removal outwardly; the former is the more frequent in small, the latter in large sequestra; but union will not result as long as the sequestrum remains between the granulations of the fragments. Since the opening at *d* may be much contracted by the development of external callus, the operative removal of the necrosed ends is often very difficult. We find, by examination with the probe, whether such sequestra in the deeper parts really existed, and if they are detached. If you suppose the sequestrum, *a a* (Fig. 56), removed from the wound, there is no obstacle to the filling of the wound with granulations and to their subsequent ossification. Such sequestra in complicated fractures are frequently the cause, not only of new exacerbations of the acute suppurative inflammation, but also of subacute and chronic periostitis, with protracted firm œdema of the extremity and annoying eczematous eruptions on the skin, as well as of long-continued bone fistulæ and ulcerations of the ends of the fragment. The action of this sequestrum combines the double effect of a foreign body and that of local or general purulent infection.

We may speak here of conditions as they exist in the bone after amputation. Imagine Fig. 56 divided transversely at the point of fracture and the lower half removed, then the condition will be just the same as after amputation. Granulations either grow directly from the wounded surface, or a portion (the sawed surface) is necrosed to a greater or less extent (Fig. 57). Let this be as it may, in the medullary cavity, as well as on the outside of the bone, a neoplasia (a half callus) is formed; this subsequently ossifies; if you examine an amputation stump several months old, you will find the medullary space in the stump of the bone closed by osseous deposits, as well as external thickening of the bone. We may here remark that the name callus is used almost exclusively for the bony new formation in fractures, while the young bony formations on the outside occurring in various ways are called "osteophytes" (from ὀστέον, bone, and φῦμα, tumor); callus and osteophytes are not then very different, but both are designations for young osseous formations.

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In considering the process of suppuration, we have left out of consideration two of the constituents of bone, namely, the periosteum and medulla. In observing the development of callus, we saw that the periosteum also had something to do with the formation of new bone. But, if, in open suppurating fractures, the suppurative inflammation spreads greatly as a result of extensive contusion, a large amount of periosteum may necrose or suppurate, and in such cases we find wide-spread *suppurative periostitis ;* the greater part of a long bone, as the tibia, may be bathed in pus. The bone thus losing its connection with the soft parts, its supply of blood is withdrawn, and from this cause there may be extensive necrosis of the bone as a result of suppurative periostitis. But these local dangers are slight in comparison to the dangers to the organism at large from these deep suppurations; we shall hereafter treat fully of these.

In the same way the medulla either of a long or spongy bone may participate in the suppuration. From what has already been said, you know that, in the course of the normal union of fracture, new bone-tissue forms in the medullary cavity, and closes it for some time. In open, suppurating fractures there is also occasionally suppuration of the medulla, that may extend more or less. Such a suppurative osteomyelitis is quite as dangerous, both for the life of the bone and for the entire organism, as suppurative periostitis. From various causes, too, it may asssume a putrid character; the larger veins of the bone, that come from the medulla, may participate in the suppuration, and this disease is the more destructive because of its deep situation; it is often first recognized at the autopsy. Purulent osteomyelitis alone may also lead to partial and even to total necrosis of a bone, the more so when combined with suppurative periostitis.

Although it was necessary to make you acquainted with all the above local complications of open fractures, I may say for your relief that they rarely occur so extensively as above described; neither total necrosis of both ends of the fracture, nor extensive purulent periostitis and osteomyelitis are frequent results of these fractures; but, fortunately, healing of the deeper parts often takes place very simply, and suppuration only continues externally.

Fracture does not always result from the action of a strong direct force on a bone; the injury may vary from contusion of the periosteum to crushing of the bone. There may have been merely compression of the periosteum, or the force may have bent the bone, which sprang back to its normal shape without breaking; but at the same time the medulla may have been greatly crushed. In the spongy substance there may be slight breaks or bends, which are never entirely dissipated, even though the cortical substance has not perceptibly changed its form. All these injuries of bones resulting from strong compression are classed as contusions of bone. Concussion of bone may result from either direct or indirect force, and be marked by ruptures of the medulla and hæmorrhages. After these injuries, pains and disturbance of function are greater than after injuries of the soft parts; a certain diagnosis of grade is often impossible at first. Occasionally concussions of bone with contusion (as in falls on the great trochanter) result in long-continued ostitis, which is not often accompanied by suppuration, but by formation of osteophytes, sclerosis, and protracted impairment of function, which in old persons may prove permanent.

Whether a traumatic inflammation leading to suppuration shall extend beyond the borders of the irritation (of the injury) depends, as in simple contused wounds, on the grade of the local infection by mortifying tissue in the wound, and later on all the circumstances that we have learned as direct or indirect causes of secondary inflammation of wounds.

[The injury to the soft parts at the time of the fracture has a great effect on the cure ; for experience shows that extensive injuries to bones, as in gunshot injuries, are very dangerous, not so much on account of the fractures as from coincident tearing and crushing of the parts around the bone. On the other hand, in osteotomies of the long bones you may separate a piece by the chisel and injure the medulla so that fat-drops will stream out with the blood, and nevertheless the wounded bone may heal without formation of a drop of pus, if the soft parts have not been injured or the wound been infected. This would show that for the bone, as well as the soft parts, it is not so much the injury as the complications that cause inflammation and suppuration. Bone really offers the most favorable conditions for healing by granulation without reaction. It is not strange that inflammation and suppuration occur in many cases of complicated fracture not treated antiseptically : they may be rendered unclean by foreign bodies at the time of injury ; moreover, the soft parts are bruised, and between the ends of the bone and all around lie blood and tissue-fluids, which are mixed with air at the time of injury and by movements of the parts, thus receiving more ferments. If we take the most favorable cases, where the soft parts and bone are capable of living and must not necessarily mortify, decomposition of the extravasations can hardly be prevented ; fluid containing cocci collects in the wound and smells bad, even the day after the injury. The more difficult the escape of the secretions through the wound in the soft parts, the more the tissue is crushed and the circulation disturbed, the more quickly will decomposition extend to the whole wound ; the decomposing matter will be absorbed and induce general septic infection, and wherever it infiltrates the tissues an intense inflammation arises, first in the soft parts, but subsequently in the medulla and periosteum. If the escape of secretion be impeded, as when thick layers of muscles cover the bone, and the wounds of the different layers do not exactly correspond, the decomposing secretion may be so compressed between the ends of the fractured bone as to be actually pressed into the medulla, and induce progressive osteomyelitis. Thus we see how important are the injuries to the soft parts in complicated fractures, and how much subsequent symptoms depend on the repair of the surrounding tissues; and that the amount of injury done to them greatly affects the prognosis. The prognosis also depends greatly on how soon after the injury the case comes under treatment: in the one case the aim of treatment would be to avoid infection, in another to arrest or modify a process of decomposition already begun. In this respect complicated fractures are like contused wounds, but the conditions are more unfavorable; the more the complication approximates an incised wound the better the prognosis.

Where the force applied has been very great, death may result in a few hours from shock or from rapid septic poisoning; the case is about the same as after contused wounds. Even when a limb is amputated at once, the patient may die in a few hours; if he lives twenty-four to forty-eight hours, it may be difficult to say whether he dies from shock, or acute septicæmia, or from both. Decomposition may be so rapid that a quantity of poison passes at once into the lymph and blood-vessels and induces sudden intoxication—so sudden that its symptoms hardly have time to develop.

Another complication that may prove fatal, either in complicated or simple fractures, is *fat-embolism* of the blood-vessels. For fat-molecules to enter the circulation, there must at some point be free fat (i. e., fat not enclosed in cells), and injured vessels through whose openings drops of fat may pass and be conveyed along by the blood. These conditions are present in any fracture, but particularly where the medulla has been extensively crushed and its vessels torn. As fat-drops entering the circulation are carried centripetally, they reach the right heart, thence pass to the lungs, where they are caught in the capillaries of the alveoli, as may be seen with the microscope; they act as mechanical emboli, no change taking place in their vicinity. Harmless as limited embolism of this sort may be, and often as it probably occurs without reaction, it is very dangerous when it takes place, not only in numerous capillaries of the lungs, but in the brain, heart, intestines, liver, kidneys, etc.; in such cases the fatty embolism may be the sole and immediate cause of death, as is shown by many published cases. Under these circumstances death may result in a few days after the injury; but this unfortunate termination is rare, and the diagnosis should be proved by autopsy to render it certainly correct.

The treatment of complicated fractures is one of the most difficult things in surgery. Years ago a complicated fracture was usually an indication for amputation, the dangers being thereby so much diminished; and by the modes of treatment then in vogue more patients were saved by amputation than by conservative treatment. Later, as open treatment of wounds and fixed dressings came into use, indications for primary amputation were limited; and conservatism was encouraged by the good results from plaster dressings, and aiding the escape of secretion by leaving the wound open, by drainage, by counter-openings, etc. Perhaps we have gone to the opposite extreme, and may now amputate too rarely; and with the idea that open treatment prevents decomposition, and that inflammation is reduced by absolute rest, conservative practice may be carried too far, and the patient be exposed to various dangers that we cannot control, and, even when healing is complete, he may have a useless, misshapen limb, that may be more of a burden than benefit.

Neither the open treatment nor fixed dressings entirely remove the dangers of acute progressive inflammations or of secondary suppurations, and their results, septicæmia and pyæmia; many patients died of these complications in spite of treatment by ice, immersion, or drainage. It is only of late, since the antiseptic treatment has been so thoroughly carried out, that the treatment of complicated fractures is really favorable. This limits the indications for amputation almost to those cases where recovery would result with a useless limb.

In the conservative treatment of complicated fractures we wish to avoid—1. Retention of the secretions of the wound; 2. Putrid decomposition in the wound, with its local and general consequences; 3. Secondary progressive suppurations in the wound. As the impulse for decomposition, by the entrance of organized exciters of putrefaction from without, is usually given at the time of injury, the chief point of treatment is disinfection of the recent wound, to get rid of germs already in and to destroy and remove all substances that favor the development of micrococci, especially the secretions from the wound. Of course, it is not always easy to fulfil this double indication. In some cases with gaping wounds and superficial division of the bone, made by cutting machines, etc., simply washing out the wound with antiseptic solutions may prevent infection and decomposition. You must learn, in the clinic, to distinguish such cases from severe injuries; in case of doubt it is better to be unnecessarily careful in disinfecting.

In most cases complicated fractures are not readily accessible to examination and treatment, the wound of the skin being too small to allow satisfactory inspection. The first thing is to determine the extent of the injury; for instance, of fracture of the femur due to concussion. The patient is brought into the hospital just as he was found, except that the leg has been fastened to an improvised splint. A cursory examination shows that there is an injury of the bone; the patient or his companions may say the bone is broken. To make an accurate examination and apply a permanent dressing it is well to anæsthetize the patient, unless this is contraindicated by his condition of acute anæmia, shock, or concussion of the brain; then remove the clothes, temporary dressings, etc.; cleanse the wound and surrounding parts with soap and warm water, or, if necessary, turpentine or ether. Then examine the fracture and skin-wound by introducing the finger (well disinfected) to determine the direction of fracture, dislocation, etc., and to remove foreign bodies, loose bone, etc. Then cleanse and disinfect the wound; for this purpose it is often necessary to enlarge the skin-wound; of course this is to be done in the direction where fewest vessels will be divided, or mostly in the long axis of the limb and the incision should give you free access to the fractured ends of the bone. Now wash the wound with a two-per-cent. solution of carbolic acid, being very careful to remove foreign bodies (earth, sand, metals, wood, rags, etc.), blood-coagula, shreds of tissue, bone, etc., but leaving adherent portions of bone. *Volkmann* advises dislocating both ends of bone outwardly through the wound, carefully cleansing them, cutting off splinters, etc. I do not consider this as always necessary, but the surfaces of the fracture should be freed from the adherent coagula and shreds of tissue.

This manipulation may start arterial hæmorrhage, or there may have been bleeding; in such cases the clots should be turned out and the source of hæmorrhage sought, the bleeding point caught with forceps and ligated with disinfected catgut or silk. Should a large artery be torn, if we cannot ligate it in the wound we should do so in the continuity, but this is rarely necessary. Bleeding from the medulla of the bone or capillaries of the soft parts may be arrested by irrigation with cold solutions of carbolic acid, elevation of the limb, or very hot water, 125° Fahr.

If the wound has been cleansed so that the injection return uncolored, you reduce the fracture by extension and counter-extension and pressure on the ends of the bone. All obstacles to exact reposition should be removed, bone splinters cut off, tendons and fasciæ between the fragments moved to one side or cut, and then sewed together. The reduction being completed, examine again with the finger for any pockets or spaces that should be drained or have counter-openings. When a counter-opening is required, make it as nearly opposite the fracture as possible; pockets should be opened at their deepest point, and the drainage-tubes led out the shortest way. Long drains, obliquely through the wound, are easily obstructed, and if not removed at the right time cause fistulæ; they should not be used. As a general rule, drainage-tubes should be as short as possible, and perpendicular to the surface.

When the drainage-tubes have been introduced, and secured by a safety-pin or suture, we should see that they act properly by injecting a two-per-cent. solution of carbolic acid and noticing if it escapes well. Now disinfect the whole wounded surface with a five- to eight-per-cent. solution of chloride of zinc, to destroy any germs that may have entered already, and to induce a thin crust on the surface that may protect it from infectious matters; this "thin crust" is a grayish-white coating, due to coagulation of the tissue-fluids, and does not interfere with healing by first intention. When the ends of the bone have been brought into position, and the drainage-tubes arranged, you may unite the wound made by incision by sutures deep and superficial; parts that have been much crushed will, of course, be left free, and the edges are not to be united so as in any way to interfere with escape of secretion or cause tension. These sutures cause as much as possible to heal by first intention. The case is now ready for dressing, and *Lister's* dressing, with plenty of gauze, may be applied, making regular compression and covering the limb for some distance around the fracture; then apply gutta-percha paper, wadding, and another bandage; outside of this *Lister's* dressing we may place splints of wood, gutta-percha, zinc, pasteboard, starch bandage, etc., or in fractures of the femur, with the antiseptic dressing, we may use *Buck's* extension apparatus. Bear in mind that the fate of the case may depend on this first dressing, as the local and general complications of compound fractures are due exclusively to infection of the wound from the entrance of ferments. If at the first dressing we succeed in fully disinfecting the wound, and removing all foreign bodies, blood-coagula, etc., the patient will be in about the same condition as after a simple fracture. The absorption of the undecomposed secretion from the wound has little effect; the wound, properly drained and brought together by the bandage, may unite mostly by first intention, without interfering with the escape of secretion. Usually between the ends of the bone, and filling the whole cavity, there is a blood-clot coming from the vessels of the medulla and the soft parts after the dressing has been applied; this prevents the entrance of air to the cavity; if this clot escapes infection from organic germs it plays the part of the coagulated fibrine between the edges of a simple incised wound; wandering cells enter it, new vessels throw out loops, and in normal cases the coagulum becomes organized and serves as the first provisional union of the fractured ends. If the first dressing has been successful, the subsequent treatment is very simple; in twenty-four hours the dressing is changed, to get rid of the portions impregnated with bloody serum; the second dressing may stay on till the fourth or fifth day, or till the secretion shows through. If all goes well, many or all of the drainage-tubes may be removed, and subsequent dressings may be left on a week or two, till the wound is all healed. If there be still some granulations, they may be treated with salves.

If the first dressing has not succeeded, or if decomposition had already begun in the wound, we cannot expect much benefit from *Lister's* dressing; on removing the first dressing you may notice that the secretion smells bad, the patient complains of pain, there is swelling about the wound, and sensitiveness to touch; pressure brings out dark, decomposing blood which has active phlogogenous and pyrogenous properties. Should you decide to attempt arresting this decomposition, the only way is to open the wound and cleanse it with a strong solution of carbolic acid or chloride of zinc, but such attempts rarely succeed. Blood-clots infiltrated in the soft parts form so many foci for development of coccobacteria; you cannot disinfect the wound perfectly because its borders have been infiltrated, and micrococci have entered the spaces between the connective tissue.

Where the first *Lister's* dressing has not succeeded, you had better relinquish it and resort to the open treatment; remove the sutures where necessary, fill the cavity with charpie wet with *Burow's* solution, and over the whole apply compresses wet with the same fluid. Sometimes even this does not answer, or you do not see the fracture till the third or fourth day, and find the whole wound decomposing, and about the fracture there is marked swelling and infiltration, then you may best try permanent irrigation with *Burow's* solution; by this you may hope to arrest the decomposition, and at least to escape the complications of septic phlegmon and secondary suppurations. In such cases the utmost attention is required to recognize at once any retention of secretion or accumulation of pus, and render them harmless by drainage or counter-openings. Of course there is no hope of healing by first intention in such cases, and we may be glad if the inflammation and suppuration remain limited to the wound, and do not spread.

When the wound is filled with granulations the permanent irrigation may be abandoned, but the drainage-tubes be left in position for a time; of course, healing goes on more slowly than under normal circumstances, and there is more danger from complications than when *Lister's* dressing is used.

Of late *iodoform* has been extensively employed in surgery, and among other cases in compound fractures. The peculiar action of iodoform on proliferating, fungous granulations, will be treated of hereafter; here it is enough to say it has a very decided antiseptic action, probably because its contact with the tissues induces continuous formation of iodine; for iodine may be detected in the secretions of patients treated with iodoform. This drug is a yellow crystalline powder, insoluble in water; it is usually sprinkled on in substance, or wounds are packed with it, *Lister's* dressing being applied over it. In compound fractures, extensive contused wounds, especially when ragged, after due preparation the cavity may be filled with iodoform, and the dressing left on till the secretion comes through. A peculiarity which renders the use of iodoform objectionable for some persons is its smell. Success with iodoform treatment depends a good deal on whether a wound is sufficiently accessible; if not you may have to resort to permanent antiseptic irrigation.

Sometimes several weeks after the injury there will be swelling, profuse suppuration, and high fever, which may be due to partial necrosis of sharp fragments; if so, the attempt should be made during anæsthesia to extract the offending bone; otherwise the necrosed fragments should be left to be pushed out by the granulations, and no unnecessary attempts at sounding should be made until the wound has become like a chronic bone fistula.

Healing of compound fractures is always tedious—it may take far more than twice as long as a simple fracture; we should not allow the patient to use the limb till manual examination shows it is firm. The disappearance of callus, and other changes, occur as in simple fractures.]

# APPENDIX TO CHAPTERS V. AND VI.

## LECTURE XVII.

1 Retarded Formation of Callus and Development of Pseudarthrosis.—Causes often unknown.—Local Causes.—Constitutional Causes.—Anatomical Conditions.—Treatment: internal, operative; Criticism of Methods. 2. Obliquely-united Fractures; Rebreaking, Bloody Operations.—Abnormal Development of Callus.

### 1.—RETARDED DEVELOPMENT OF CALLUS AND FORMATION OF A SO-CALLED FALSE JOINT—A SO-CALLED PSEUDARTHROSIS.

UNDER some circumstances, which we do not always sufficiently understand, a fracture is not consolidated after the lapse of the usual time; indeed, it may not consolidate at all, but the seat of fracture may remain painless and movable, which of course impairs the function of the limb, even to the point of entire uselessness. A short time since, a strong farmer-boy, with simple subcutaneous fracture of the leg without dislocation, entered the hospital; as usual, a plaster-bandage was applied and renewed in fourteen days. Six weeks after the fracture the dressing was removed altogether, in the expectation that union had taken place; but the point of fracture was still perfectly movable, nor could any callus be felt. I here tried the simplest remedy in such cases, I narcotized the patient, and then rubbed the fragments strongly together till crepitation could be distinctly perceived; then I applied another plaster-dressing, and on removing this in four weeks found the fracture tolerably firm. I placed the patient in a fracture-box, and, without placing any bandage on the leg, had its anterior surface painted daily with strong tincture of iodine. After this had been continued a fortnight, the fracture was perfectly firm; the patient now stood with the aid of crutches, and in a short time was dismissed cured. I know of two other cases from the practice of colleagues, where simple fractures in very healthy young persons did not consolidate, but formed pseudarthroses. Such occurrences are to be regarded as very rare; usually there is some peculiar cause, such as disease of the bone, that induces false joint. There are certain fractures of the human skeleton which from various causes very rarely unite by bony callus; among these, are intracapsular fractures of the neck of the femur, neck of the humerus, and fractures of the olecranon and patella. When fractured transversely the two latter bones separate so far that the osseous substance formed on the two ends cannot meet, so that only a ligamentous union can take place between these two parts of bone. When fractured within the capsule the head of the femur has, it is true, a supply of blood through a small artery which enters it through the ligamentum teres, but this source of nutrition is very slight, consequently the production of bone from the small fragments is slight. In fracture of the head of the humerus within the capsule, in the rare case of part of the head being entirely detached from the rest of the bone, this portion of bone will receive no supply of blood, and will act as a foreign body; its union can scarcely be expected. In the above examples, we regard non-union so much as the rule that we do not usually call them cases of pseudarthrosis. But I wish to show you that there may be purely local causes that predispose to pseudarthrosis; among these especially belongs complete loss of large pieces of bone, after the removal of which, in open fractures, there may be so large a defect that it will not be again filled by new bone-tissue. Protracted suppuration with ulcerative destruction, and extensive detachment of the ends of the fragments, may also lead to pseudarthrosis. Moreover, the treatment is occasionally blamed; too loose a dressing, or none at all, and too early motion, are occasionally accused. On the other hand, it has been asserted that too continued application of cold, the simultaneous ligation of large arteries, and, lastly, too tight a dressing, may interfere with proper development of bony callus. All this alone does not necessarily lead to pseudarthrosis, but may act as a second cause when the general conditions of nutrition in the organism predispose to it.

Of the general predispositions and bone diseases, the following may be mentioned as disposing to pseudarthrosis: bad nutrition, debility from repeated losses of blood, specific diseases of the blood, such as scorbutis, or cancerous cachexia. Of the diseases of the bones, it is chiefly osteomalacia, atrophy of the cortical substance, with enlargement of the medullary cavity, in which, as already mentioned, in certain stages there is not only decided fragilitas ossium, but in which also the chances for reunion are slight. I have stated all this, because it is generally accepted, although, on sharp critical examination, some of the above-mentioned predisposing causes for pseudarthrosis are of very different value, while the significance of others is entirely doubtful. In the same way it is a common belief that fractures are not consolidated in pregnant females. This is not true in all cases; I have myself seen numerous fractures unite in pregnant women, only once hardening of the callus was delayed a few weeks in a fracture of the lower end of the radius, which was recognized late, as might also occur in women not pregnant, or in men.

The abnormity of the healing process in case of pseudarthrosis is not due to the non-formation of callus, but to the failure of ossification in the new formation. The substance connecting the fragments becomes a more or less rigid connective tissue, by which the ends of the bone are held more or less closely together. If the fragments lie so close that they come in contact on motion of the limb, a cavity with smooth walls, filled with sero-mucous fluid, forms between them in the uniting tissue; and, on the fractured ends, cartilage has been found, so that there was, in fact, a sort of new joint. This does not, however, occur very often, but in most cases we have simply a firm connecting mass, which sinks directly into the fragments like a tendon. When such a pseudarthrosis is in a small bone, such as the clavicle, or one of the bones of the forearm, the disturbance of function is always bearable; but, if it be located in the arm, thigh, or leg, of course there must be considerable impairment of function. In some cases it is possible, by suitable supporting apparatus, to give the limb the necessary firmness; in other cases we cannot do this at all, or only incompletely, so that for a long time attempts have been made to cure this disease by operation, that is, by inducing ossification. Before passing to the methods used for this purpose, we must mention the attempts made to prevent false joint, and to cure it, when once established, by internal remedies. Preparations of lime are chiefly used for this purpose. Phosphate of lime was given internally in the shape of powder; lime-water was given in milk, but without much benefit. Of the lime given in this way, little is absorbed, and, of this superfluous lime taken into the blood, much was excreted through the kidneys, so that the pseudarthrosis had little good from it. We may expect more from general regulation of diet, and prescribing articles of food that contain lime. Residence in pure country air, and milk-diet, are to be recommended; but you must not expect too much from these remedies, especially in a fully-formed false joint that has existed for years. In a recently-published and very interesting work by *Wegner*, it is shown, by a number of experiments, that by continued administration of small doses of phosphorus the formation cf callus about fractures is particularly luxurious and hard, as well as that in growing animals the portion of bone formed during the administration of phosphorus is unusually dense and hard, and very rich in chalky salts. These experiments would lead us to try phosphorus in patients with pseudarthrosis, especially in the earlier stages; of course, we should be very careful of this remedy, which may be so dangerous when carelessly used. The local remedies all aim at inducing inflammation in the ends of the bone and parts around, because experience shows that most inflammations in the bone, especially subcutaneous traumatic ones, induce formation of bone in their immediate vicinity. The remedies employed vary very greatly. We have already mentioned the proposals to leave the limb without dressing, so as to avoid interfering with the formation of the external callus by pressure, also the rubbing together of the fragments, and painting with iodine; with the same view (viz., of irritating the fragments), we may apply *blisters* and the *hot iron* to the part of the limb corresponding to the fracture. By the following remedies we act more on the intermediate ligamentous tissue: long, thin *acupuncture-needles* are passed into the ligamentous band, and left there for a few days to excite irritation; we may connect the ends of two of these needles with the poles of a galvanic battery, and pass an electrical current as an irritant. This proceeding is called *electro-puncture;* it is little used. We may also pass a thin, small tape, or several threads of silk (a so-called *seton* or a strong *ligature*), through the ligamentous tissue, and leave it there till there is free suppuration around it. The following operations attack the bone more directly; they are quite numerous: For instance, a narrow but strong knife is passed as deep as the fracture, and the ligamentous tissue is shaved first from the end of one fragment, then from the other, without enlarging the skin-wound. This is called the *subcutaneous bloody freshening* of the fragments. Or we may make an incision down to the bone, dissect out the two fragments, perforate them close to the fractured end, and pass a sufficiently thick lead wire through the perforations, twist the ends together, so as to approximate the fragments, or else, after making an incision, we may saw off a thin piece from each fragment, and treat the resulting wound like an open fracture; and to this operation, *resection of the fragments*, we may add the application of a *suture of the bone.* The following operation originates with *Dieffenbach:* Corresponding to the ends of the fragments he makes two small incisions down to the bone, then he perforates the ends of the bone close to its borders, and with a hammer drives *ivory pegs*, of suitable thickness, into the perforations. The consequence is, a formation around these foreign bodies of new bone, which, when extensive enough, as it may always be made in the course of time by repeating the operation, causes firm union. I will here mention that, when extracted in a few weeks, these ivory pegs look rough and corroded at the points where they were in contact with the bone, while the perforation in which they lay is mostly filled with granulations; occasionally the pegs are not removed; the openings through which they were introduced heal. This proves absolutely that *dead bone*, among which ivory is to be classed, *may be dissolved and reabsorbed by the growing osseous granulations*. We shall hereafter have frequent occasion to return to this much-contested question, which is very important in some bone-diseases; we have already spoken of the theoretical causes of this reabsorption (p. 197). *B. v. Langenbeck* has modified this operation of *Dieffenbach* by using metal screws instead of ivory pegs; immediately after the operation he fastens these screws to an apparatus, which keeps the fragments immovable. After all these operations, a suitable dressing must be applied to keep the fragments firm.

The modes of operation in pseudarthrosis, of which I have only mentioned the principal ones, are, as you see, quite numerous; and, if the results of treatment corresponded to the number of remedies, this would belong to the most curable class of diseases. But in medicine you may generally take it that, with the increase in number of remedies for a disease, their value decreases. Easy and certain as some forms of pseudarthosis are to cure, others are just as difficult; nor are all the different methods suited to the same case. In the first place, the operations vary greatly as to danger, being much more dangerous in limbs with thick soft parts, especially in the thigh, than in others; and, as may be readily supposed, the non-bloody operations are less dangerous than the bloody; those made with a small wound less so than those with larger. As regards efficacy and certainty, I consider the introduction of a *bone suture* and *resection* as those which, even in the worst cases, give proportionately the quickest results, but which still have all the elements of danger of a fracture complicated by a wound. The treatment with ivory pegs is less dangerous, except in the thigh, where every false joint is dangerous, and I think it would accomplish the object in most cases, if the operation were repeated often enough. I have seen good results from this treatment, and from *Von Langenbeck's* screw apparatus, as well as from the bone suture.

In pseudarthrosis of the thigh the question may seriously be asked, if we should not prefer amputation at the point of the false joint (which is of favorable prognosis) to any other dangerous or doubtful operation. This question only the peculiarities of the individual case can decide. In some cases the safe aid of a suitable splint apparatus, made by a skilful instrument-maker, is preferable to any operation.

## 2.—OBLIQUELY-UNITED FRACTURES.

Although, with the progress made in the treatment of fractures, it is now rare for union to occur in so oblique a direction as to render the limb entirely useless, still, cases from time to time arise where, in spite of the greatest care of the surgeon, in fractures with open wounds, dislocation cannot be avoided, or else, from carelessness or great restlessness of the patient and loose application of the dressings, a considerable obliquity in the position of the fracture remains. In many cases this is so slight that the patients do not care to get rid of the deformity; improvement of the position would only be desired in cases where, from considerable obliquity or shortening of a foot or leg, the movements are decidedly impaired. There are various means by which we may greatly improve or entirely get rid of these deformities. If, during the process of union, we notice that the fragments are not exactly coapted, we may undertake the adjustment at any time in simple subcutaneous fractures. If, in an open fracture, obliquity of the fragments has taken place under the first dressing, I strongly urge you not to try to rectify it before the wound has healed; you would thus break up the deeper granulations, and the severest inflammation might again be excited. In fractures that have long suppurated, the callus long remains soft, so that you may always subsequently accomplish a gradual improvement in position by properly padding the splints first in one place, then in another, or perhaps by continued extension with weights. If the fracture be fully consolidated in an oblique position, we have the following remedies for its improvement:

1. Correction by bending the callus, by *infraction;* for this purpose we anæsthetize the patient, and with the hands attempt to bend the limb at the point of fracture; if we succeed in so doing, we apply a firm dressing with the limb in the improved position. This method, so free from danger, can only be successful while the callus is still soft enough to be bent; hence it can only be done soon after the fracture.

2. Complete breaking up of the ossified callus. This also may sometimes be done by the hands alone, but frequently other mechanical means will have to be resorted to. For this purpose various apparatuses have been constructed, such as lever and screw machines of considerable power; one of the most terrible bears the name of "dysmorphosteopalinklastes." All these apparatuses should only be used with the greatest care, so as not to cause too much bruising and consequent necrosis of the skin at the point where the machine is applied on which the limb rests. For the not unfrequent obliquely-united fractures of the thigh, the *forced extension* of *A. Wagner* (by th

apparatus of *Schneider* and *Menel*, which we also employ for reducing old dislocations) has been resorted to with success. The following illustration will fully explain the mechanical effect of this extension: If you have a bent rod, and let a strong man take hold of each end and draw, the rod will break at the point where it is bent most. If a new fracture of the thigh has been caused by indirect force at the bent part, and the fragments be adjusted in a straight position, you apply a plaster-dressing at once while the limb is still held in the machine. As far as our present experience goes, this method appears to be entirely free from danger, but only suited for the thigh; in a case of very angular union of a fracture of the leg, where I advised this treatment, the break caused by the extension was not in the old seat of fracture, but near it.

3. The bloody operations on the bone, of which there are two in use, are more dangerous; the first of these is the ***subcutaneous osteotomy*** of *B. v. Langenbeck*. This consists in making a small incision down to the bone at the bent part, introducing a medium-sized gimlet through this opening and perforating the bone, without, however, piercing the soft parts on the opposite side; then draw out the perforator, and pass a small, fine saw through the perforation, and saw the bone transversely, first to one side, then to the other, till you can break the rest of the bone with your hand; now the bone is to be straightened and the injury treated as a complicated fracture. This operation has only been done on the leg, but, so far as I know, always with good result. It may also be done by not making the adjustment till suppuration begins, and the callus has thus been softened and partly reabsorbed. For *V. Langenbeck's* instruments we may advantageously substitute fine chisels, as recommended by *Gross*, for dividing the callus from a small exposed portion of the bone.

4. Lastly, we may employ the method of *Rhea Barton*, which consists in exposing the bone by a large incision through the skin at the point of curvature, and sawing out a wedge-shaped piece in such a way that the broad part of the wedge shall correspond to the convexity, the point to the concavity of the abnormal curvature of the bone. This method also shows good results.

On the whole, the non-bloody are to be preferred to the bloody methods, if they do not cause too much contusion; but the latter are less dangerous than breaking up fractures with strongly-contusing apparatuses.

If the deformity, especially of a foot, be so great, in different directions, that none of the above methods offer much prospect of cure, we may have to resort to amputation in some cases.

In some few cases the callus is abnormally thick and extensive, just as happens in cicatrices of the skin and nerves. Do not be too hasty about operating in such cases, for slow subsequent reabsorption usually takes place in every callus. The removal of such callus masses could only be effected with chisel or saw, and I should be unwilling to decide on such an operation.

[Operations for osteotomy should be made with strict antiseptic precautions, and these are not dangerous where the chisel can be used instead of the saw: we escape the bone-dust.

It seems as if oblique fractures of the tibia, without much displacement, formed a large part of the cases of delayed formation of callus; many of the cases have had very little local reaction, and have been placed in fixed dressings immediately after the injury. Another accident is retarded ossification of callus. Recovery may seem perfect and dressings may be left off, and after a day or two you may find shortening or angular deformity of the limb. In such cases the callus has not properly ossified, and the bone behaves as a rachitic bone. After a time complete consolidation results, although the deformity may persist. Sometimes pseudarthrosis is attributed to bad treatment, the dressing has been too loose, the limb not properly splinted, or it has been moved too soon, etc., etc. These assertions are all uncertain, as are the claims that proper development of callus may be impeded by application of cold, ligation of large arteries, paralysis of the extremities, or by a bandage too firmly applied.

Among the local means of treatment may be mentioned painting the skin over the part with tincture of iodine; this induces plastic infiltration of tissue extending to the periosteum and bone; blisters and the cautery act in the same way. Another means, sometimes efficacious, is constriction of the extremity by binding on a thick compress of wadding above the fracture so as to compress the veins of the limb and cause swelling; after a few days' repetition of this, swelling of the ends of the fragment shows formations of callus.]

# CHAPTER VII.

## *INJURIES OF THE JOINTS.*

Contusion.—Distortion.—Opening of the Joint, and Acute Traumatic Articular Inflammation.—Variety of Course, and Results.—Treatment.—Anatomical Changes.

Hitherto we have studied injuries of simple tissue-elements; now we must occupy ourselves with more complicated apparatuses.

As is well known, the joints are composed of two ends of bones covered with cartilage; of a sac frequently containing many appendages, pockets, and bulgings; the synovial membrane, which is classed among the serous membranes; and of the fibrous capsule of the joint with its strengthening ligaments. Under some circumstances, all these parts participate in the diseases of the joint, so that at the same time we may have disease of a serous membrane, of a fibrous capsule, as well as of cartilage and bone. The participation of these different parts varies exceedingly in intensity and extent; but I may state at once that the synovial membrane plays the most important part, and that the peculiarity of joint-diseases is chiefly due to the closed and irregular form of the synovial sac.

First, a few words about crushing and *contusion of the joint.* If one receives a heavy blow against the joint, it may swell moderately; but in most cases, after a few days of rest and applications of lead-water or simple cold water, the swelling and pain subside, and the functions of the joint are restored. In other cases, slight pain and stiffness remain; a chronic inflammation develops, which may lead to serious disease, of which we cannot at present speak more fully. If we have a chance to examine a moderately-contused joint, the patient having died perhaps of a serious injury received at the same time, we shall find extravasations of blood in the synovial membrane, and even blood in the cavity of the joint itself; in these contusions without fracture the effusions of blood are rarely so extensive that the joint is tensely filled with blood; but this may occur. This condition is called *hæmarthron* (from αἷμα, blood, and ἄρθρον, joint). If a joint that has swollen greatly just after an injury remains painful for some time, and feels hot, a somewhat more active antiphlogistic treatment is indicated. This consists in the application of leeches, regular envelopment of the joint in wet bandages, causing moderate compression, and in applying an ice-bladder to the joint. As a rule, inflammation of this grade may be readily relieved, although chronic diseases and a certain irritability of the joint that has been injured not unfrequently follow. It is very important to determine whether the crushing of the joint be accompanied by fracture or fissure of the end of the bone, in which case, it would be necessary to apply a plaster-dressing, and give a guarded prognosis as to the future usefulness of the joint. If the continued application of cold increases the pain, make inunctions of mercurial ointment, and apply moist, warm compresses covered with gutta-percha and wadding.

A form of injury peculiar to joints is *distortion* (literally, twisting). This is an injury that occurs especially often in the foot, and which is commonly called "turning the foot." Such a distortion, which is possible in almost any joint, consists essentially in a tension, too great stretching and even partial rupture, of the capsular ligaments, with escape of some blood into the joint and surrounding tissue. The injury may be very painful at the time, and its consequences are not unfrequently tedious, especially if the treatment be not rightly conducted. Usually abstraction of blood and cold are resorted to in these cases also, but with only temporary benefit. It is much more important to keep the joint perfectly motionless after such injuries, so that, if any of the ligaments be ruptured, they may heal and acquire their previous firmness. The simplest way of attaining this object is by applying a firm dressing, such as the plaster-bandage, with which we may permit the patient to go about, if it gives him no pain. After ten, twelve, or fourteen days, according to the severity of the injury, we may remove the dressing, but renew it at once if the patient has pain on walking. It may sometimes be necessary to wear this dressing three or four weeks. This appears a long time for such an injury; but I can assure you that, without the application of a firm dressing, the consequences of these sprains often continue for months, at the same time the danger of subsequent chronic inflammation of the joint is increased. Hence you must not promise too speedy a cure, and must always treat these, often apparently insignificant injuries, conscientiously and carefully.

[Besides immobilization an excellent remedy for the relief of pain and restoration of function after slight sprain is rubbing and kneading the part immediately after the injury, a procedure used by the Hindoos and Grecians. This is called *massage*, and consists in rubbing the skin all over the injured part, as far as the joint above, with oil or some fatty substance; the hand of the rubber should also be well greased, so as to prevent friction on the skin. Then rub at first lightly, and gradually with increasing pressure, from below upward toward the body, so that the fingers applied below the injured joint are carried up to the next joint, as if you would press a fluid from the periphery toward the centre of circulation. It has been shown by experiment that granular coloring-matter (as cinnabar, India-ink, etc.) injected into the knee of a rabbit may be made to enter the lymphatic vessels and spaces of the connective tissue of the thigh within a few minutes by massage. The same is true of pathological products, blood-extravasations, etc.

There is no doubt that by massage we may induce rapid absorption of blood and inflammatory swelling; the pain, at first severe, disappears during the massage. The resolvent effects are most decided in the four or six hours after the injury; later, when there is acute inflammation present, it seems less reliable, but when the acute inflammatory swelling has passed it may be tried more energetically. Later we shall treat of the employment of this method in chronic affections of the joints.

Unfortunately, in spite of the most careful treatment, chronic inflammations often follow, which not only prove tedious but in the course of years destroy the joint by fungous disease; this is especially apt to occur in children and feeble persons of scrofulous habit.]

### OPENINGS OF THE JOINTS, AND ACUTE TRAUMATIC ARTICULAR INFLAMMATIONS.

In now passing to wounds of the joint, we make an immense spring as regards the importance of the injury. While a contusion and sprain of the joint are scarcely noticed by many patients, the opening of a synovial sac, with escape of synovia, even if the wound be not large, always has a serious effect on the function of the joint, and is not unfrequently dangerous to life. Here, again, we have the difference between subcutaneous traumatic inflammations and those which open outwardly, of which we spoke when on the subject of contusions, and which we also saw in subcutaneous and open fractures. Moreover, in the joints, we have closed irregularly-shaped sacs, in which the pus, once formed, remains, and, besides inflammation of the serous membranes, may result in very tedious processes, but in its acute state often has a bad effect on the general health of the patient.

I think the quickest way to describe the process will be to give you a few examples. We are here speaking only of simple punctured, incised, or cut wounds, without complications from sprains or fractures, and choose as our example the knee-joint; at the same time we must remark that injuries of this joint are regarded as the most severe. A man comes to you, who, in cutting wood, has received a wound half an inch long, near the patella, and which has bled but little. This may have happened some hours before, or even the previous day. The patient pays little attention to the wound, and only asks your advice about a proper dressing. You inspect the wound, find that from its position it corresponds to the knee-joint, and around it you may perhaps see some serous, thin, mucous, clear fluid, which escapes in greater quantities when the joint is moved. This will call your attention particularly to the injury; you examine the patient, and learn from him that, immediately after the injury, there was not much bleeding, but a fluid like white of egg escaped. In such cases you may be certain that the joint has been opened, otherwise the synovia could not have escaped. In small joints the escape of synovia is so slight as to be scarcely noticeable, hence, in injuries of the finger-joint, and even of the ankle, elbow, and wrist, it may for a time be doubtful whether the wound has penetrated the joint or not. [While a person with simple fracture of the thigh is compelled by pain and loss of function to regard himself as a patient, one with a wound of the knee-joint may go around with little difficulty and be unaware of the serious nature of his injury. In no class of wounds do such serious results come from neglect, and great is the responsibility of the surgeon who does not recognize the danger of the case at the start.

Treatment will depend on whether the wound is very recent or has been exposed to the air. Examination should first be made for foreign bodies, for hæmorrhage, and as to the extent. If you can hope that there has been no infection and that little blood has entered the joint, disinfect the wound and parts around, and try to induce union as rapidly as possible, closing the wound by deep sutures; then apply a *Lister's* dressing and make even compression.

An important point in treatment of all wounds of joints is absolute rest; this you should ensure by proper splints or a plaster dressing, which should be worn till the wound in the soft parts has closed, and the joint is entirely free from inflammation. Under this treatment the wound may heal by first intention. If the wound has become infected, leeches, cups, ice, etc., do little good; if it has not, they are superfluous. In the joint there is usually an increase of synovia, which causes swelling; but the even compression may induce its rapid absorption; even in a week the joint may have its natural size. At first the patient will have tension and heat and be feverish, but, when the swelling subsides, feels well and free from pain. Even in such cases it is well to leave the fixed dressing on for four weeks, or even longer, if there is any swelling or sensitiveness, and then to begin motion very gradually; the joint has become stiff and the muscles atrophied; at the first passive motions we usually feel slight grating of the articular surfaces; this soon disappears under methodical and careful gymnastics and massage; in addition you may employ tepid baths, cold douches, frictions with spirits of camphor, ammonia, etc., and sometimes moist warmth. In favorable cases motion is soon perfect.

But, if the wound was infected at first, or if there was considerable hæmorrhage into the joint, and air has entered it, the case is entirely different; even if the wound of the soft parts was insignificant and the edges had closed so that the joint-wound is not perceived, for the first two or three days all seems to go smoothly, the fourth or fifth day you may find swelling of the joint, the skin over it is red and hot, the sutures have cut into the flesh, and thin pus escapes from the wound. The skin-wound may be superficially closed, and on removing the sutures the edges gape and synovia mixed with pus may flow from the joint. If there has been hæmorrhage and entrance of air into the joint, in the course of twenty-four hours there is usually intense local and general reaction, the joint is tense and painful, the patient feverish; if you open the adhesions the third or fourth day, a dark, badly-smelling fluid composed of synovia, pus, and blood-coagula, escape. These are the most dangerous cases; if not quickly recognized and treated, there is speedy decomposition in the joint.

To take an example of a less acute process: A man has been stuck in the knee with a butcher's knife, and has had no proper treatment; he may have limped around for some days in spite of the pain, but toward the end of the week this becomes so severe that the slightest motion or touch is unbearable; the patient with both hands and the other leg holds it quiet; about the joint it is red, hot, and the knee and leg are swollen; there is fever, with evening exacerbation, loss of appetite, and emaciation. It may be that after the escape of some turbid synovia the wound has adhered again, or else there is continued flow of sero-mucus gradually changing to pus, or else swelling and fluctuation of the joint; the great pain, increased temperature, œdema of the thigh, etc., indicate intense arthritis. In such cases, if the limb be not fixed, the joint becomes flexed even to a right angle.

The cause of this flexion of inflamed joints is probably that the sensitive nerves of the synovial membrane induce contraction of the flexors by reflex irritation. The explanation that flexion occurred because joints hold most fluid when between flexion and extension, has been disproved; flexion may occur in joint inflammations without collection of fluid, and is often absent in chronic, painless dropsy of the joints. The high pressure necessary in *Bonnet's* experiments to induce flexion of the joint probably never occurs in living patients. At all events, observation teaches that acute *painful* synovitis is most apt to cause flexion, and on the other hand that pain decreases if, during anæsthesia, the joint be straightened, and if fixed in this position, pain gradually ceases.

If proper treatment be not adopted, the fluid in the joint increases rapidly; the tension is greater, pain unbearable, till finally the skin-wound is too small for the escape of the secretion, the articular capsule is perforated by pus which escapes into the surrounding tissue. Spontaneous cure may thus occur, the pus about the joint quickly reaching the surface, and the abscess closing up gradually. In such cases the final result is tolerably good if the acute synovitis has not caused too much destruction; the fever subsides as soon as the pus escapes, the local symptoms of inflammation moderate, and finally the perforations and original wound heal; but stiffness of the joint may remain. If the faulty position be not rectified, the joint may become fixed in flexion, the ligaments, tendons, and fasciæ atrophy, harden, and may impair the use of the limb. This result is the more probable the longer suppuration continues, and the deeper synovitis has affected the parts.

But this result is not the most serious or most frequent. The rule rather is that, when suppuration has once attacked the joint and has not been properly treated, it steadily progresses; the capsule is attacked, and in spite of the open wound it ulcerates, and pus escapes into the parts around the joint. In such cases there is usually a deep, indistinctly fluctuating tumor; pressure upon which, even at a distance from the joint, evacuates slowly through the wound a thin purulent fluid. Part of the synovia may escape and continue to secrete clear serum; this is particularly apt to occur in the knee, part of the capsule being shut off from the rest by the swollen membrane, as in the blind sac under the tendon of the quadriceps and between the patella and femur; but, if the whole cavity be attacked, it is just in these spaces that pus is most persistently retained.

Even where pus has not escaped into the surrounding tissue, abscesses (periarticular) may form in the soft parts; probably from products of inflammation from the joint being carried by the lymphatics into the periarticular connective tissue, and there inducing inflammation and suppuration; later, the tissue between these abscesses and the joint may be destroyed, and communication be established. In the neighboring sheaths of the tendons suppuration creeps along, at various places perforations occur; the whole limb is filled with pus, which, on pressure, appears at the various openings; destruction of the capsule and ligaments renders the limb abnormally movable—especially in the hinge-joints do we notice lateral motion; but the patient has lost the power of active motion of the joint, the muscles are infiltrated or have suppurated. Of course, with such severe local processes, the general condition is much affected. From the first the patient has had fever; with evening exacerbations to 103°, loss of appetite and sleep, and continued pain; in three weeks he would scarcely be recognizable. If suppuration extends to the soft parts, sheaths of the tendons, etc., repeated chills will occur, the face is sunken, mind dull, the patient complains little, talks disjointedly and drowses; the tongue becomes dry and coated, speaking and swallowing are interfered with; at night there is delirium or restlessness. Even in this stage recovery would be possible if the suppuration would cease, the fever abate, and the process become chronic. But this rarely happens. Patients are usually so exhausted that, even when the local affection takes a favorable turn, they die of marasmus. But most cases die of pyæmia with pulmonary abscesses, or from profuse dysentery, or with encephalitis and suppurative meningitis.

To avoid the unfavorable results of a penetrating wound of the joint, you should prevent or limit suppuration and decomposition of the discharges. In case of a joint-wound complicated by presence of foreign bodies or considerable hæmorrhage in the joint, this should be opened freely by one or two long incisions to give free access; these incisions should be in the long axis of the limb, and made with as little injury to the soft parts as possible. Formerly it was not considered enough to open the joint, a resection was made; now this would not be done unless the ends of the bone were broken, as by a gunshot-wound, or a partial resection might be necessary to permit escape of secretions. After arresting the hæmorrhage, while kneading and moving the joint, you wash it out with quantities of a two-per-cent. solution of carbolic acid, introduce drainage-tubes, and close the wound by sutures; then apply a *Lister's* dressing and a splint, or permanent extension. If suppuration has already begun when the joint comes under treatment, or if secretions have accumulated in spite of drainage-tubes, etc., *Lister's* dressing will do little good; in such cases try permanent irrigation with *Burow's* solution, but apply it very freely, and it had better be done the first time during anæsthesia. Open the joint freely, and wherever there is a pocket make an opening. Often you will find suppurating granulations on the inner surface of the synovial sac; these should be scraped off, then rub the surface with a dry, clean sponge. Open all collections of pus around the joint, introducing a flexible probe into the deepest pockets and cutting down on its point, of course avoiding large vessels, nerves, etc.; these abscesses also may be scraped out. When possible, *Esmarch's* bandage should be previously applied. When the cavities have all been attended to, apply exact compression, to prevent bleeding when the elastic is removed, then keep the limb elevated half an hour. The dressing should be removed in twenty-four hours, when it is usually so filled with pus and blood that it may be removed without arousing hæmorrhage; the gaping wounds should again be washed out, drainage-tubes introduced when necessary, cavities filled with charpie soaked in acetate of alumina, and the limb covered with compresses wet with the same; the limb should be put in a splint, laid on a rubber cloth, two or more drainage-tubes projecting from the dressing are connected with an irrigator, and the whole complicated wound may thus be constantly washed by a stream of antiseptic fluid (*Burow's*). To secure the entrance of drainage-tubes into the deep pockets, you may pass through them lead or iron wires, so you can give them any direction. If properly applied, permanent irrigation may induce great change in the wound, even in one day. It usually arrests at once decomposition of pus; the temperature falls, pain subsides, the quantity of pus decreases; swelling and infiltration about the joint lessen. If the disease has not gone so far as to destroy the articular ligaments, the joint may recover with normal motion; to ensure this, irrigation should be continued as long as there is suppuration. If opening and drainage of the joint was attended to at first, we may certainly expect perfect cure.

In spite of the above treatment of penetrating wounds of joints, which gives results that a few years since would have appeared wonderful, cases requiring amputation do sometimes occur. If the suppuration cannot be arrested, or the patient has not come under our care till he had chills, suppuration of the soft parts, and continued fever, then the only hope of saving the patient is to amputate through the sound tissue. Even this is often ineffectual, general purulent infection having already occurred, with metastatic abscesses of the internal organs, or the patient being too much exhausted. But amputation has the advantage of removing at once the focus whence the infection proceeds. The immediate effect of this operation is often wonderful, the temperature, which for weeks may have been 103° or 104°, may sink at once to normal, or even below; the general condition improves, the dry, brown tongue becomes moist and clean, appetite returns, the patient sleeps, delirium disappears, and in two or three days he may be scarcely recognizable. The wound usually heals rapidly, unless we may have been unable to operate entirely in sound tissue. Sometimes apparently hopeless patients are saved by amputation.

Modern treatment of penetrating wounds of joints, as of compound fractures, has greatly changed the prognosis, as well as the statistics; not only do many cases now recover that formerly died, but perfect recovery, with free motion of the joint, is almost the rule in ordinary cases. Formerly a penetrating wound of a joint either led to immediate amputation or resection, or, if treated expectantly, and after escaping many dangers, the patient recovered with a limb at all useful, the result was considered a triumph; while now it is almost wrong for a case of joint-wound that comes under treatment early not to heal by first intention and without danger to life. Such favorable cases did occur before *Lister's* method was used, and may now result without treatment, but it is more by accident than due to the surgeon.

In speaking of traumatic arthritis we have said nothing of the finer pathological changes in the inflamed joint; so let us return and examine the histological processes as they have been studied on the dead body, on amputated limbs, and by experiments on animals. In inflamed joints the disease affects chiefly, and at first exclusively, the synovial membrane; unless this has been carefully dissected, we consider it much thinner than it really is; in most places it is thicker and more succulent than pleura or peritonæum, and is separated from the fibrous capsule of the joint by a loose, subserous, and sometimes very fat, layer of cellular tissue, so that you can detach the synovial sac of a knee-joint to the cartilages. It consists of connective tissue, whose surface is covered with pavement endothelium, and near the surface is a capillary network; according to *Hueter*, these membranes have no lymphatic vessels, while there are plenty in the sub-synovial tisssue. This is surprising. *Tillmans* confirmed this observation in rabbits and dogs, but in oxen found numerous lymphatics, both in the superficial and deep layers of the synovia.

The surface of the synovia shows, especially at the sides of the joint, numerous tufts; these contain rather complicated capillary loops. Synovial share with other serous membranes the peculiarity of secreting a quantity of serum when irritated; at the same time the vessels are dilated and become tortuous near the surface; the membrane loses its smooth, glistening look, and becomes dull, yellowish, then reddish and velvety. This change is primarily due to the excessive diapedesis of red blood-corpuscles which escape with the serum; but subsequently to the numerous newly-formed blood-vessels which, looped and crowded, press to the surface, and to the fine tufts, which give the velvety look. In most cases there is a fibrinous deposit, a so-called pseudo-membrane like that formed on the pleura and peritonæum.

Microscopic examination of the synovial membrane in this state shows that its whole tissue is densely infiltrated with cells, which collect so on the surface that the tissue seems to consist of small round elements, the more superficial of which exactly resemble pus-corpuscles. In the immediate vicinity of the distended vessels the cells are particularly plenty, which may be due to white blood-cells wandering from the vessels; red corpuscles also seem to escape. The pseudo-membranes are entirely composed of small round cells united by fibrinous substance. The connective tissue of the membrane has partly lost its striation and assumed a gelatinous consistence, so that it greatly resembles the intercellular substance of granulation tissue. The fluid of the joint gradually becomes turbid and purulent, and contains pus-corpuscles in gradually-increasing quantities. Still later the whole surface of the synovia becomes so vascular that to the naked eye it looks like a spongy, finely-granular surface.

The condition of the synovial membrane at first most resembles that of mucous membranes in acute catarrh. So long as there is merely superficial suppuration, without softening of the tissue (ulceration), the membrane may return to the normal state; but if pseudo-membranes have formed and the parenchyma is inflamed and suppurates, the only result is formation of cicatrix.

There may be escape of pus from the joint into the subcutaneous cellular tissue, and this is apt to occur at special points which are predisposed by their anatomical conformation; but there may also be independent suppurations about the joint not connected with it, from reabsorption of phlogogenous matter from the joint. Peri-articular abscesses come either with acute or chronic abscesses from the same cause, reabsorption of irritant matter; this is also seen exceptionally in adjacent lymphatic glands, which swell, become painful, and exceptionally suppurate; there may also be lymphangitis. Cartilage does not participate till late in the process; its surface becomes cloudy, and if the process be very acute it disintegrates into molecules or large portions become necrosed; it becomes detached from the bone by suppuration going on between them; the changes in the cartilage are chiefly passive, a sort of maceration such as occurs in the cornea in severe blenorrhœa or diphtheria. There are scarcely any two parts of the body presenting such analogy as the conjunctiva in its relation to the cornea and the synovial membrane in its relation to the cartilage.]

# LECTURE XVIII.

Simple Dislocations; Traumatic, Congenital, Pathological Luxations, Subluxations.—Eticlogy.—Difficulties in Reduction, Treatment; Reduction, After-Treatment.—Habitual Luxations.—Old Luxations, Treatment.—Complicated Luxations.—Congenital Luxations.

## SIMPLE DISLOCATIONS.

By a dislocation (*luxatio*), we understand that condition of a joint in which the two articular ends are entirely, or for the most part, thrown out of their mutual relations, the articular capsule being generally partly ruptured at the same time; at least, this is almost always the case in *traumatic luxations*, i. e., in those that have occurred in a healthy joint as a result of the application of force. Besides these, we distinguish *congenital*, and *spontaneous* or *pathological luxations*. The latter result from gradual ulcerative destruction of the articular extremities and ligaments, since there is no longer the natural opposition to muscular contraction; we shall speak of this hereafter, as it essentially belongs among the results of certain diseases of the joints. At the end of this section we shall say something about congenital luxations. At present we shall speak only of traumatic dislocations. We occasionally hear also of *subluxations;* by this expression we imply that the articular surfaces have not separated entirely, so that the luxation is incomplete. By *complicated* luxations we mean those accompanied by fractures of bones, wounds of the skin, or ruptures of large vessels, or nerves, or all of these. You must also observe that it is customary to designate the lower part of the limb as the part luxated; as for instance at the shoulder-joint, not to speak of a luxated scapula, but of dislocation of the humerus; at the knee-joint, not of luxation of the femur, but of the tibia, etc.

Dislocations generally are rare injuries; in some joints they are so rare that the whole number of cases known is scarcely half a dozen. It is said that fractures are eight times as frequent as dislocations; it seems to me that even this is too large a proportion for dislocations. The distribution of luxations among the different joints varies very greatly; let me show you this by some figures: According to *Malgaigne's* statistics, among 489 dislocations there were 8 of the trunk, 62 of the lower and 419 of the upper extremity, and among the latter there were 321 of the shoulder. Hence you see that the shoulder is a very favorite joint for dislocations, which is readily explained by its construction and free mobility. Dislocations are more frequent among men than women, for the same reasons that we have already shown fractures to be more frequent in men.

As inducing causes for dislocations, we have external applications of force and muscular action; the latter are rare, but cases have been observed where dislocations were caused, in epileptics, for instance, by muscular contractions. As in fractures, the external causes are divided into *direct* and *indirect*. For instance, if one gets a luxation by falling on the shoulder, it is due to direct force; the same luxation might occur indirectly by a person with outstretched arm falling on the hand and elbow. Whether a dislocation or a fracture will result, depends chiefly on the position of the joint and the nature of the cause; but much also depends on whether the bones or the articular ligaments give way the more readily; for instance, by the same manœuvre on different dead bodies we may sometimes cause fracture, sometimes dislocation. As in fractures, there are numerous symptoms of luxation, of which some may be very noticeable, and are the more so the sooner we see the case, and the less the displacement of the articular ends is hidden by inflammatory swelling of the superjacent soft parts. The altered form of the joint is one of the most important and striking symptoms, but which only leads quickly and certainly to a diagnosis when the eye has been accustomed to readily recognize differences from the normal form. Correct measurement with the eye, accurate knowledge of the normal form, in short, some taste for sculpture and sculptural anatomy, so-called artistic anatomy, are here very useful. If there is very slight change of form, even the most practised will not be able to dispense with a comparison with the opposite side, and hence I earnestly urge you, if you would avoid error, always to expose the upper or lower half of the body, as the case may be, and to compare the two sides. You may best follow with the eye the direction of the apparently displaced bone, and if this line does not strike the articular cavity accurately, there will most probably be a dislocation, if there be not a fracture, close below the articulating head of the bone, which must be determined by manual examination. The lengthening or shortening of a limb, its position to the trunk, the distance of certain prominent points of the skeleton from each other, often aid us in making at least a probable diagnosis very quickly. Another symptom perceptible to the sight is ecchymosis of the soft parts, or suggillation. This is rarely distinct at first, because the blood, escaping from the torn capsule only gradually, perhaps not for several days, rises near the skin and becomes visible; in some cases the effusion of blood is so inconsiderable that it is not perceived. The symptoms given by the patient are, pain and inability to move the limb normally. The pain is never so great as in fractures, and only appears on attempting to move the limb. In some cases, patients with luxations may perform some motions with the limb, but only in certain directions, and to a very limited extent. Manual examination must finally settle the question in most cases; it must show that the articular cavity is empty, and that the head of the bone is at some other point, at one side, above or below. If the soft parts be considerably swollen, this examination may be quite difficult, and the aid of anæsthesia is often necessary for a correct diagnosis, especially if the exhibitions of pain and the motions of the patient interfere. On moving the extremity, which we find springy or slightly movable, there is occasionally a feeling of friction, an indistinct, soft crepitation. This may result partly from rubbing of the head of the bone on torn capsular ligaments and tendons, partly from the compression of firm blood-coagula. Hence, in such varieties of crepitation, we should not at once conclude on a fracture, but be urged to more careful examination. Fractures of certain parts of the articular ends, with dislocation, are most readily mistaken for luxations. And formerly the mode of expression on this point was not exact, for displacements about the joint, combined with fractures, and caused entirely by them, were also termed luxations. At present we distinguish these fractures within the joint, with dislocations, more sharply from luxations proper.

Should you be in doubt as to whether the case is one of dislocated articular fracture or of luxation, you may easily decide the question by an attempt at reduction. If such a dislocation is readily reduced by moderate traction, but at once returns when you leave off the traction, it is a case of fracture; for a certain art is necessary to the reduction of a dislocation, and, when once reduced, it does not readily recur, although there are exceptions to this rule.

A contusion and sprain of the joint may also be mistaken for luxation, but this error may be avoided by careful examination. Old traumatic luxations may sometimes be mistaken for dislocations caused by contraction. Lastly, in paralyzed limbs, where there is at the same time relaxation of the articular capsule, the joint may be so very movable that in certain positions it will look as if dislocated. In these cases, also, the history of the case and careful local examination will lead us to a correct conclusion.

Regarding the state of the injured parts immediately after the injury, in cases where there has been a chance to examine them, it has been found that the capsule of the joint and the synovial membrane are torn. The capsular opening is of variable size; occasionally it is a slit like a button-hole, sometimes it is triangular, with more or less ragged edges; ruptures of muscles and tendons immediately around the joint have also been observed. The contusion of the parts varies greatly, as does also the effusion of blood. The head of the bone does not always remain at the point where it escapes from the ruptured capsule, but in many cases it is higher, lower, or to one side, as the muscles attached to it contract and displace it. It is important to know that we must frequently bring the luxated head of the bone into a different position before we can carry it back through the opening in the capsule into the articular cavity.

Occasionally, by some accidental muscular action, the dislocation is spontaneously reduced. In the shoulder, especially, this has been observed several times. But such spontaneous reductions are very rare, because there are usually certain mechanical obstructions to the reduction, which must be overcome by skilful manipulation. These hinderances consist partly in contraction of the muscles, by which the head of the bone may be caught between two contracted muscles; another far more frequent obstacle is a small capsular opening, or its occlusion by the entrance of the soft parts. Lastly, certain tensions of the capsular or strengthening ligaments may hinder the reposition of recent traumatic luxations.

In treating a luxation it must first be skilfully reduced, and then means be employed for restoring the function of the injured limb. We shall here only speak of the reduction of recent dislocations, by which we mean those that are at most eight days old. The most favorable time for reducing a dislocation is immediately after the injury; then we have the least swelling of the soft parts, and little or no displacement of the luxated head of the bone; the patient is still mentally and physically relaxed from the accident, so that the reposition is not unfrequently very easy; later, we shall often have to facilitate the operation by resorting to anæsthetics to remove the opposition of the muscles. Regarding the proper manœuvres for the reduction, we can say but little in general terms, for this of course depends entirely on the mechanism of the different joints. Formerly, it was a general rule, for the reduction of dislocations, that the limb should be brought into the position in which it was at the moment of the dislocation, so that by traction the head of the bone might be replaced as it had escaped. This rule is only important in a few cases; at present, in the different dislocations we are more apt to resort to very different motions, such as flexion, hyper-extension, abduction, adduction, elevation, etc. Usually, the surgeon directs the assistants to make these motions, and himself pushes the head of the bone into place when it has been brought before the articular cavity.

Frequently the surgeon alone can accomplish the reduction. I have often thus reduced a dislocation of the thigh over which various colleagues, aided by muscular laborers, had worked in vain for hours. In these cases, every thing depends on correct anatomical knowledge, and you may readily understand that in a certain direction you may not unfrequently slip the head of the bone into place with very little force, while in another position it might be entirely impossible. When the head of the bone enters the articular cavity, it occasionally causes a perceptible snap; but this does not always occur; we are only perfectly assured of successful reposition by the restoration of normal mobility.

If we do not succeed alone, or with a few assistants, we have various aids, by applying long slings to the limb, and having several assistants draw in one direction. This traction, which of course must be opposed by a counter-extension of the body, must be regular, not by starts. If we do not succeed, even in this way, we call in the aid of machinery to increase the power. For this purpose various instruments were formerly employed, such as the lever, screw, ladders, etc. Now the multiplying pulleys, or *Schneider-Menel's* extension-apparatus, is almost exclusively used. The multiplying pulleys, an instrument that you already know from your studies in physics, for increasing power, and which is greatly resorted to in mechanics, are used as follows: One end of the rope is fastened to a strong hook in the wall, while the other is applied to the limb by straps and buckles. Counter-extension is made on the body of the patient, so that it shall not be moved by the extension. An assistant draws on the pulleys, whose power of course increases with the number of rollers employed. *Schneider-Menel's* apparatus consists of a strong gallows, to the inner side of one post of which is attached a movable windlass, which may be turned by a handle and held by a toothed wheel; over this windlass runs a strap which is attached by a hook to a bandage applied around the luxated extremity. In luxation of the lower extremity the patient lies on a table placed lengthwise between the posts of the gallows, or for reduction of an arm he may be seated on a chair placed the same way; the counter-extension is made by straps by which the patient is fastened to the other post of the gallows. Both of these apparatuses have certain advantages, but both are troublesome to apply. In your practice you will have little to do with them, as they are almost exclusively employed in old dislocations whose treatment is more rarely undertaken in private practice than in hospitals and surgical clinics.

At present, when we undertake this forcible reduction, it is always under the influence of anæsthetics. To produce complete relaxation this anæsthesia must be very profound, and, as the chest is often covered with straps and girdles for counter-extension, the anæsthetic must be very carefully employed to avoid dangerous results. But there are also other dangers which were known to the older surgeons, who did not use chloroform. These are as follows: If the patient is tried too long with these powerful remedies, he may suddenly collapse and die; moreover, the limb may become gangrenous from the pressure of the straps, or there may be subcutaneous rupture of large nerves and vessels, and consequent paralysis, traumatic aneurism, extensive suppuration, and other dangerous local accidents. The results of pressure from the appliances may best be avoided by applying a moist roller-bandage from below upward, and fastening the straps over this. Since a regular pressure is thus made over the entire limb, the pressure of the appliance close above the joint does not prove so injurious. The time during which we may continue these forcible attempts at replacement should be at most half an hour; if we do not succeed in this time, we may be pretty certain of not doing so at all. If we wish to try further in such cases, we should resort to some other method. Until recently, we had no measure of the force that could be used without danger, and had to content ourselves with estimating it. It seems scarcely possible, by the above means, to tear out an arm or a leg; but not long since this did occur in Paris, and in a case where only manual extension was employed! Generally, the straps tear sooner, or the buckles bend. Subcutaneous ruptures of the nerves and vessels would scarcely be caused in a healthy arm by regular traction on the whole extremity; but they may tear, when adherent to deep cicatrices, and are so atrophied as to have lost their normal elasticity. If, under such circumstances, the conditions could always be accurately appreciated beforehand, we should frequently entirely abstain from attempts at reduction; for, in such cases, rupture of a nerve or vessel may follow attempts at reposition with the hand, and we cannot refer the accident to the machinery. An instrument has been invented, by whose aid the force employed in extension may be measured. This instrument should be inserted in the extension-apparatus, and shows the force employed in weight, as is customary in physics. According to *Malgaigne*, we should not go above two hundred kilogrammes with this dynamometer; but such directions are of course only approximative.

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If the reduction has been accomplished, the main point has certainly been gained, but some time is still required for full return of the function of the limb. The wound in the capsule must heal, for which purpose perfect rest of the joint for a longer or shorter time is requisite. After reposition there is always moderate inflammation of the synovial membrane, with a slight effusion of fluid into the joint, and the latter remains for a time painful, stiff, and unwieldy. If reduction has closely followed the injury, the joint must first be kept per fectly quiet; it is surrounded with moist bandages, and cold compresses are applied; the swelling is rarely so great as to demand other antiphlogistic remedies. In the shoulder-joint after ten to fourteen days we begin passive motion and continue it till active movements can be made and the arm is fully useful; frequently, it is many months before movements are quite free, and elevating the arm is the last motion to return. In other joints that have less mobility, active movements may be permitted much sooner; thus they are restored especially early in the elbow and hip-joints, and in the latter joints we may permit attempts at motion the earlier, because there luxations do not so readily recur.

If active motions be permitted too soon after reduction of a dislocation, especially in those joints where dislocation readily recurs, as in the shoulder and lower jaw, and if the luxation recurs once or several times before the capsular opening has healed, occasionally the capsule does not heal completely, or there is so much distensibility of the capsular cicatrix that the patient only has to make a careless motion to luxate the part again. Then we have the state called *habitual luxation*, a very annoying state, especially in the lower jaw. I knew a woman who had a dislocation of the jaw and did not take care of herself long enough afterward, so that it soon returned and had to be reduced again; the capsule was so much stretched that, if, in eating, she took too large a morsel of food between the back teeth, she at once luxated the jaw; she accustomed herself to the manœuvre of slipping it into place, so that she could do it with the greatest facility. Such an habitual luxation of the shoulder may occur in the same way. I have seen a young man, who, when gesticulating violently, had carefully to avoid raising his arm quickly, as he almost always dislocated it by this motion; such a state is very annoying, and is difficult to cure; recovery would only be possible by long rest of the joint, but patients rarely have inclination or patience for this treatment. It is well for such patients to wear a bandage that will prevent lifting or throwing back the arm too much; if the luxation be avoided for a few years, it will not recur so readily.

If a simple dislocation be not recognized and reduced, or if, for various reasons, we cannot reduce it, a certain amount of mobility is nevertheless restored, which may be considerably increased by regular use. From the relation of the head of the bone to adjacent bony processes, and from displacement of muscles, it may be readily understood that, for purely mechanical reasons, certain motions will be impossible, while others may approximate the normal mobility. But, if the movements be not methodically restored, the limb remains stiff, the muscles become atrophied, and the limb is of little use. The anatomical changes in the joint and parts around are as follows: the extravasated blood is reabsorbed; the capsule folds together and atrophies; the head of the bone rests against some bone in the vicinity of the articulating cavity; for instance, in luxation of the humerus inward against the ribs under the pectoralis major, the soft parts about the dislocated head become infiltrated with plastic lymph and transform to cicatricial connective tissue, which partly ossifies, so that a sort of bony articular cavity again forms, while the head is surrounded by a newly-formed connective-tissue capsule. In the cartilage of the head of the bone, the following changes visible to the naked eye occur: the cartilage becomes rough, fibrous, and grows adherent to the parts on which it lies, by a cicatricial, firm connective tissue. In the course of time this adhesion becomes very firm, especially if not disturbed by movements. The metamorphosis of cartilage to connective tissue, followed microscopically, takes place as follows: the cartilage-tissue divides directly into fine filaments, so that the tissue acquires first the appearance of fibrous cartilage, then of ordinary cicatricial connective tissue, which unites with the parts around and receives vessels from them. The surrounding muscles, as far as they are not torn, lose a large part of their filaments, partly from molecular disintegration, partly from fatty metamorphosis of the contractile substance; subsequently, new muscular filaments form in these muscular cicatrices.

This is what we call an old luxation, and it is in such cases especially that the above methods of forcible reduction are employed. The question, how long a luxation must have existed before its reposition is to be considered impossible, cannot be answered since the introduction of chloroform, and is to be differently answered for the various joints. Thus, dislocations of the shoulder may be reduced after existing for years, while those of the hip-joint two or three months old are reduced with difficulty. The chief obstacle lies in the firm adhesions of the head of the bone in its new position, and in the loss of contractility of the muscles, and their degeneration to connective tissue. Another question is, whether, when such old dislocations are reduced, we attain the desired effect on the function, especially in the shoulder. Imagine that the small articulating cavity is filled by the atrophied capsule, and that the head of the bone has lost its cartilage, then, even if we succeed in bringing the head to its normal position, restoration of function may still be impossible, and I can assure you, from my own experience, that the final result of a very tiresome and long after-treatment in such cases does not always repay the patience and perseverance of the patient and surgeon. In such cases, the result will scarcely be more favorable than if the patient tries, by methodical exercise, to make his limb as useful as possible in its new position, which it may have occupied for months or years. We may facilitate this exercise by breaking up the adhesions about the head of the bone, by rotating it forcibly while the patient is anæsthetized. If, as occasionally happens in shoulder-dislocations, the head of the bone in its abnormal position so presses on the brachial plexus as to cause paralysis of the arm, if reduction be impossible, it may be advisable to make an incision down to the head of the bone to dissect it out and saw it off, i. e., to make a regular resection of the head of the humerus. I have seen a case where, in complete paralysis of the arm after a luxation of the humerus downward and inward, decided improvement of the function of the arm was attained by the above operation, although there was not complete recovery of the paralysis.

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## COMPLICATED DISLOCATIONS.

A dislocation may be complicated in various ways; most frequently with partial or entire fracture of the head of the bone, which is difficult to cure, and in which reposition is often only partly successful; in treatment, attention must always be paid to the fracture; i. e., a dressing must be worn till the fracture has united. At the same time it is advisable to renew the dressing frequently, say every week, and to apply it in a different position each time, so that the joint may not become stiff. Nevertheless, we cannot always succeed in attaining perfect mobility, so that I can only advise you in your practice always to give a doubtful prognosis in such cases.

Another complication is a coincident wound of the joint. For instance, the broad articular surface of the lower epiphysis of the humerus or of the radius may be driven out of the joint with such force as to tear through the soft parts and skin, and lie exposed.

Of course the diagnosis is easy in such cases; reposition is accomplished according to the above rules, but we still have a wound of the joint; and we are liable to all the chances spoken of under wounds of joints, so that for the prognosis, the varieties of the possible results and the treatment, I refer you to what has already been said (p. 224). Of course, it is worse when there is an open fracture through the joint; here we can neither expect rapid closure of the wound nor restoration of the function of the joint, and we run all the dangers that threaten complicated open fractures and wounds of joints. The decision as to what must be done in such cases is easy, when there is at the same time considerable crushing or tearing of the soft parts; under such circumstances, primary amputation must be done. If the injury of the soft parts be not great, we may sometimes hope for a cure by suppuration, with a subsequent stiff joint; but, as experience shows, this is always a dangerous experiment. According to the principles of modern surgery, in such cases we avoid amputation by dissecting out and sawing off the fractured articular ends of the bones so as to make a simple wound. This is the regular *total resection* of a joint, an operation concerning which very extensive observations have been made during the last twenty years, and of which modern times is justly proud; by its means many limbs have been preserved, which, according to the old rules of surgery, should unhesitatingly have been amputated.

In regard to their danger, these resections vary greatly according to the joint on which they are made, so that it is difficult to make any general remarks about them. But, in a subsequent section (in the treatment of chronic fungous diseases of the joints), we shall study this very important point more carefully; what has been said will give you a general idea of a resection of the joint.

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## CONGENITAL LUXATIONS.

Congenital luxations are rare, and we must distinguish them from *luxationes inter partum acquisitæ*, i. e., those that have resulted at birth from manœuvres in extracting the child, and which are merely simple traumatic luxations which may be reduced and cured. Although congenital luxations have been observed in most of the joints of the extremities, they are particularly frequent in the thigh, and not unfrequently occur on both sides at the same time. The head of the bone stands somewhat above and behind the acetabulum, but in many cases it can readily be replaced. As a rule, the disease is first noticed when the child begins to walk. The most noticeable symptom is a peculiar wabbling gait, which is caused by the head of the bone standing behind the acetabulum so that the pelvis inclines forward, and also because in walking the head of the thigh moves up and down; there is never any pain. To examine the child more accurately, you may unclothe it entirely and watch its gait; then lay it on the back and compare the length and position of the extremities. If the luxation be one-sided, the luxated limb will be shorter than the other, and the foot turned inward; if you fix the pelvis, you may often reduce the dislocation by simple traction downward. The anatomical examination of such joints has led to the following results: not only is the head of the bone out of the socket, but the socket is irregularly formed—too shallow; later in life, in adults, it is greatly compressed and filled with fat; when the ligamentum teres exists, it is abnormally long; the head of the bone is not properly developed; in some cases it is not half as large as normal; the articular cartilage is usually completely formed, the capsule very large and relaxed.

Under such circumstances, you may understand that it is exceedingly difficult, in most cases impossible, to effect a cure. If the head be only partially developed, the upper border of the acetabulum absent, and the capsule enormously distended, how shall we restore the normal conditions? As to the causes of this malformation, the most varied hypotheses have been advanced; the opportunity has never occurred of studying it in the embryo. There is an arrest of development from some cause. It is assumed that these disturbances followed previous pathological processes in the fœtus, and the most probable hypothesis is that, in very early embryonal life, the joint was filled with an abnormal quantity of fluid, and so distended as to induce rupture or at least great dilatation of the capsule. *Roser* thinks that abnormal intra-uterine positions may give rise to these luxations.

Cure of this state has been attempted in those cases where direct examination has shown the existence of a tolerably-developed head. In such cases the luxation has been reduced, and attempts made to preserve the normal position of the thigh by aid of dressings or bandages—the child being kept quiet for a year or more. The result of this treatment, which requires great patience on the part of the surgeon and parents of the child, is shown by experience to be only partially satisfactory, as after this treatment there has only been an improvement of the gait, but rarely a perfect cure; and, when you read in orthopedic pamphlets of the frequent cure of congenital luxations, you may be sure that in most cases there have been errors of diagnosis, or there is intentional deception.

Congenital luxations of the thigh are never dangerous to life, but, since they are accompanied by a change in the centre of gravity of the body, in the course of time they have an effect on the position and curvature of the vertebral column; this, and a limping, wabbling gait, are the only inconveniences they cause. There can only be a hope of successful treatment in very early youth; but, as the surgeon cannot promise a successful result in less than one to three years, few patients are put under treatment.

I will here mention a very rare occurrence, which I have only met with once. In certain movements the tendon of the long head of the biceps brachii may slip out of its groove and hang on the border of the greater or lesser tubercle; then the arm stands fixed in a slightly abducted position. If we hold the shoulder-blade steady and relax the tendon by slowly raising the arm, then by slightly rotating the arm we can easily slip the tendon into place; the pain ceases at once, and all motions are free. For this luxation to occur, the fascia-like membrane which covers the sulcus must tear or be much relaxed. The former is improbable; where the latter is the case, the accident readily recurs. Some persons have the covering of the sulcus, in which the tibialis posticus muscle lies, so relaxed that they can voluntarily luxate this tendon and let it snap into place with an audible sound.

[Cases where the tendon of the quadriceps femoris and patella may be voluntarily luxated and snapped back into place are probably less rare. The translator, among other cases, has had one patient 18 months old who could do this at pleasure, and when irritated about any thing would snap his patella even if his leg were firmly held. This leg having been placed in a plaster dressing, he began the same performance with the other leg. Then both legs were left free, and after some weeks, when his general health and temper improved, the phenomenon ceased.]

[In diagnosis you may exclude luxation when the region of the joint is filled and there is no pathognomonic depression where it should occur; for instance, in fracture of the humerus without luxation the normal rounding of the shoulder will be present, and on palpation you can discover the head of the humerus in the glenoid fossa.

When luxations have existed a long time, the articular cavity becomes filled with neoplastic cicatricial tissue; this, with the fixation of the dislocated head of the bone, hinders reduction.

In attempting reductions of old dislocations we must bear in mind the liability to rupture of atheromatous arteries, or of those that have become adherent to cicatrices; the latter complication is especially dangerous with large venous or nerve trunks.

Abnormal mobility of a joint after luxation is not solely due to continuance of the rupture of the capsule or stretching of the cicatrix in it, for it does not enclose the ends of the bone so closely that it would prevent luxation; this is done by atmospheric pressure, and also by the articular ligaments and tendinous aponeuroses woven into the capsule. In every complete dislocation these are torn; like all fibrous tissues they heal with difficulty, and if movements are begun before consolidation is complete the parts separate and the joint remains abnormally movable.

When a dislocation has existed a long time, and the cavity has not been filled up, it becomes too small for the head of the bone, just as the orbit decreases in size after removal of an eye, on account of reduced pressure on one side.

When diminished motion is due to adhesions about the joint, these may be broken up during anæsthesia by forced rotation of the head of the bone.

In irreducible luxations the question may arise as to opening the joint; under our present antiseptic methods this operation would not be dangerous, but it is rarely indicated, for recent dislocations can almost always be reduced, and, when they cannot, even opening the joint does not aid a great deal.

When fracture occurs with luxation we ordinarily delay trying to reduce the latter till the former has united; of course, if the reduction can be readily done it should be first attended to.

Among the dislocations should be classed a peculiar injury, luxation of the semilunar cartilages of the knee; this is very rare unless there are other changes in the joint, so that its occurrence is actually doubted; but it does occur, and comes as an habitual luxation. Usually it is the inner cartilage which shoves forward, so that the inner condyle of the femur, instead of sliding on the cartilage, slips behind it. The displacement usually occurs on forced flexion of the joint, the toes at the same time turning outward. The symptoms are, fixation of the knee in imperfect extension with slight external rotation of the foot; if we try to rotate the knee, there are resistance and pain, the knee may be flexed to a right angle, the cartilage may be felt at the anterior border of the joint.]

# CHAPTER VIII.

## GUNSHOT-WOUNDS

### LECTURE XIX.

Historical Remarks.—Injuries from Large Missiles.—Various Forms of Bullet-Wounds.—Transportation and Care of the Wounded in the Field.—Treatment.—Complicated Gunshot-Fractures.

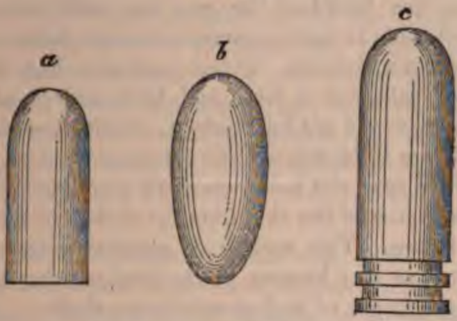
In war many injuries occur that are to be classed among simple incised, cut, punctured, and contused wounds; gunshot-wounds themselves must be classed with contused wounds; but they have some peculiarities that entitle them to separate consideration, in doing which we must briefly come in contact with the domain of military surgery. Since fire-arms were first used in warfare (1338), gunshot-wounds have received special attention from surgical writers, so that the literature on this subject has become very extensive; of late, indeed, military surgery has been made almost a separate branch, which includes the care of soldiers in peace and war, and the special hygienic and dietetic rules which are so important in barracks, in stationary and field hospitals, also the clothing and food. Although the Romans, as was mentioned in the introduction, had surgeons appointed by the state with the army, in the middle ages it was more common for every leader of a troop to have a private doctor, who, with one or more assistants, very imperfectly took care of the soldiers after a battle, and then usually went on with the army, leaving the wounded to the care of compassionate people, without the commander or the army taking the responsibility.

It was not till standing armies were formed that surgeons were detailed to certain battalions and companies, and certain (still very imperfect) rules and regulations were made for the care of the wounded. The position of military surgeon was, in those days, very gnoble, and such as we do not hear of now; for, even in the time of the father of Frederick the Great, the army surgeon was publicly flogged if he permitted one of the long grenadiers to die. At that time, when the troops marched to meet the enemy at a parade-step, the movements of the army were very tedious and slow; the large armies had immense trains; for instance, in the Thirty Years' War, the lancers carried along their wives and children in innumerable wagons; hence, in the medical arrangements pertaining to the train, there was no necessity for greater facilities of motion. The tactics started by Frederick the Great required greater mobility of the heavy trains, which, however, was only practically carried out in the French army under Napoleon. As long as a small province remained the seat of war during a whole campaign, a few large hospitals in neighboring cities might suffice; but, when armies moved about rapidly and had a fight now here now there, it became necessary to furnish more movable, so-called field hospitals, not far from the field of battle, and which could be readily moved from place to place. These ambulances, or flying hospitals, are the idea of one of the greatest of surgeons, *Larrey*, of whom we have already spoken. As I shall shortly tell you what is done with the wounded from the time they are injured till they enter the general hospital, I will here dismiss this subject, and only mention some of the many excellent works on military surgery. Especially interesting, not only medically but historically, are the somewhat lengthy "Memoirs of Larrey," in which I especially recommend to you the Egyptian and Russian campaigns. These memoirs contain all Napoleon's campaigns. Another excellent work we have in English literature, *John Hennen's* "Principles of Military Surgery;" and in German, besides many other excellent works, we have "The Maxims of Military Surgery," by *Stromeyer*, which is composed chiefly of experiences in the Schleswig-Holstein War; and, lastly, "Principles of General Military Surgery, from Reminiscences in the Crimea and Caucasus, and in the Hospital," by *Dr. Pirogoff.*

Wounds caused by large missiles, such as cannon-balls, grenades, bombs, shrapnel, etc., are partly of such a nature that they kill at once, in other cases tear off whole extremities, or so shatter them that amputation is the only remedy. The extensive tearing and crushing caused by these shot do not differ from other large crushed wounds caused by machinery, which unfortunately now so often occur in civil practice.

Musket-balls used in modern warfare differ in some respects: while the small copper bullets with which the Circassians shoot are scarcely larger than our so-called buckshot, large, hollow, leaden bullets were used in the late Italian War; these were much larger than the old-fashioned ones, and were particularly dangerous, because they readily broke upon striking a bone or tense tendon. Besides these, the solid round and conical bullet are used, but their effects do not

Fig. 58.



a, Chassepot; b, needle-gun; c, mitrailleuse-projectiles. Natural size.

differ much. The Prussian long bullet, which is held in the cartridge of the needle-gun, is a solid bullet of the form and size of an acorn. You must not think that the projectile, as found in the wound, has the same shape as when put in the gun; but it is changed in form as it comes out of the rifles of the gun, and is also flattened in the wound, so that we often find it a shapeless mass of lead, which scarcely shows the form of the projectile. We shall now briefly consider the various injuries that may be caused by a bullet; in doing which, we shall naturally confine ourselves to the chief forms.

In one set of cases the bullet makes no wound, but simply a *contusion* of the soft parts, accompanied by great suggillation and occasionally by subcutaneous fracture. According to recent authorities, simple subcutaneous fractures are not very uncommon in war. These injuries are caused by spent bullets, i. e., such as come from a long distance and have not force enough to penetrate the skin; such a bullet, striking over the liver, may push the skin before it and make a depression in or a rupture of the liver, and then fall back without producing an external wound. Like injuries are caused by bullets striking the skin at a very oblique angle. Firm bodies, such as watches, pocket-books, coins, leather straps on the uniform, etc., may also arrest the bullet. These contused wounds, which, especially when affecting the abdomen or thorax, may prove very dangerous, have always excited the attention of surgeons and soldiers; formerly they were always referred to the so-called "wind of the ball," and it was thought that they were caused by the bullet passing very close to the body. The idea that injuries could be caused in this way was so firmly established, that even very well-informed persons worried themselves in trying to explain theoretically how they resulted from the wind of the ball. One said that the air in front of and near the bullet was so compressed that the injury was due to this pressure; another thought that, from the friction in the barrel of the gun, the bullet was charged with electricity, and could in some unknown manner cause contusion and burning at a certain distance. If the conclusion that the whole idea of the wind of balls was a fable had been arrived at sooner, these fantastic theories would not have arisen. Contusions from spent and oblique bullets are to be treated like other contusions.

In the second case, the bullet does not enter the soft parts deeply, but carries away part of the skin from the surface of the body, leaving a gutter or furrow. This variety of gunshot-wound is one of the slightest, unless, as may happen in the head, the bone is grazed by the bullet, and portions of lead remain in the skull.

The third case is where the bullet enters the skin without escaping again; the bullet enters and generally remains in the soft parts; it makes a tubular wound. Various other foreign bodies may be carried into these wounds, such as portions of uniform, cloth, leather, buttons, etc.; a bone may also be splintered, and the splinters driven into the wound and tear it. After perforating the skin and soft parts, the bullet might rebound from a bone and fall out of the same opening, so that you would not find it in the wound, in spite of there being only one opening. The wound that the bullet makes on entering the body is usually round, corresponding to the shape of the ball; its edges are contused, occasionally bluish-black, and somewhat inverted. These characteristics hold in the majority of cases, but are not absolute.

The fourth and last case is where the bullet enters at one point and escapes at another. If the course of the wound is entirely through the soft parts, and the bullet has carried in no foreign body, the point of exit is usually smaller than the entrance, and is more like a tear. If the bullet has struck a bone and driven bone-splinters or other foreign body before it, the point of exit is occasionally much larger than the entrance; there may also be two or more points of exit from bursting of the bullet into several pieces or from several splinters of bone. Lastly, splinters of bone may make openings of exit like those from a bullet, while the latter, or part of it, remains in the wound. Too much value has been attached to the distinction of the openings of entrance and exit; this is only important in forensic cases, where it may be desirable to know from which side the bullet came, as this may give a clew to the author of the injury. The course of the bullet through the deep parts is occasionally very peculiar; its course is sometimes deviated by bones or tense tendons and fasciæ, so that we should be greatly mistaken in supposing that the union of the points of entrance and exit by a straight line always represented the course of the bullet. In this respect, the encircling of the skull and thorax is most peculiar: for instance, a bullet strikes the sternum obliquely, but without sufficient force to perforate this bone; the bullet may run along a rib under the skin to the side of the thorax, or even to the spinal column, before escaping again; from the position of the points of entry and exit, we might suppose the bullet had passed directly through the chest, and be greatly astonished when such patients come, without any difficulty of breathing, to have their wound dressed.

The complication of gunshot-wounds with burns by powder, such as results from shooting at close quarters, rarely occurs in war. It is not rare in cases of accidents from careless handling or bursting of fire-arms, or from blasting, and may cause the greatest variety of burn. The burnt particles of powder often enter the skin and heal there, giving it a bluish-black appearance for the rest of life. More of this in the chapter on burns.

In gunshot injuries, there is said to be scarcely any pain; the rapidity of the injury is such that the patient only feels a blow on the side from which the bullet comes, and does not for some time perceive the bleeding wound and actual pain. There are numerous examples where combatants have received a shot, especially in the upper extremity, without knowing it till told by some one, or having their attention attracted by the flow of blood.

In gunshot, as in contused wounds, the bleeding is usually less than in incised and punctured wounds; but it would be a great mistake to suppose that arteries which have been shot through do not bleed. On the contrary, many soldiers never leave the battle-field, having died from rapid hæmorrhage from large arteries. When one has seen a fully-divided carotid, subclavian, or femoral artery bleed, he will know that in a very short time the loss of blood will be so great that the only hope of safety lies in immediate aid; so that a hæmorrhage of two minutes' duration from one of these arteries is certainly fatal. But arteries, even as large as the radial, often bleed but little. The first surgeons who gave us descriptions of gunshot-wounds called attention to this point.

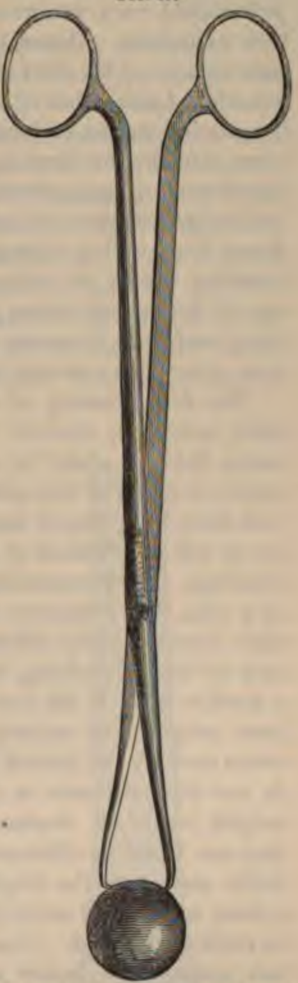
Before passing to the treatment of gunshot-wounds, I would briefly picture to you the transportation of and first aid offered to the wounded in battle. For the first aid there are usually established certain temporary places for dressing the wounded, in some sheltered place close behind the line of battle, usually in rear of the batteries; these are designated by white flags. The wounded are first brought to this spot, either by soldiers or by a trained ambulance corps. Of course, those wounded slightly or in the upper extremities walk to the spot. The ambulance corps has proved so efficient in late wars that it will certainly be more trusted to in future. It is composed of nurses trained to bring the wounded from the field, and, when necessary, to give them temporary aid, as in arresting bleeding from arteries and wounds, etc. They have been trained to carry a patient between two of them, either without other support, or on an improvised litter. For this latter purpose they usually carry a lance and a piece of cloth longer and broader than the body. The lances are passed through hems along the sides of the cloth, and a barrow is thus made; bayonets or swords may be used as provisional splints for supporting a limb that has been badly shot. The wounded are thus brought to the dressing-place, and the first dressings are applied; these remain on till the patient reaches the nearest field-hospital. At the same time hæmorrhage must be securely arrested, and injured limbs so arranged that transportation may do no harm; bullets, foreign bodies, and loose splinters of bone near the surface, should be removed, if it can be done quickly and readily. Limbs that have been entirely crushed by large shot should be at once amputated, if a dressing cannot be so applied as to render transportation possible. The chief object of this dressing-place is to render the wounded transportable; hence it is not proper to do many or tedious operations there. From the great pressure of the constantly-increasing throng from the front, only the most important cases can be attended to here, and *Pirogoff* is right, though it seems cruel, when he says the surgeons should not exhaust their strength on the mortally wounded and the dying. But, if possible, every patient, when carried to the field-hospital, should receive a short written account of what was found at the first examination; a card, containing a few words, thrust into one of his pockets is enough. The chief point is to tell whether the ball has been extracted, whether a wound of the breast or abdomen is perforating, etc., which will save time to the surgeon at the hospital and pain to the patient. Part of the ambulance corps has the further duty of placing the wounded properly in wagons for further transportation, under direction of the surgeon. For this purpose there are special ambulances, constructed most variously, which take some patients lying down, others sitting up. There are rarely enough of these, and it is often necessary to use common wagons, covered with hay, straw, etc. These wagons convey the wounded to the next field-hospital, which is established in a neighboring city or town; for it the largest attainable rooms should be taken. School-houses, churches, or barns, may be seized, although the latter are the best. In these places beds are prepared with straw, a few mattresses, and bedclothes. Surgeons and nurses await anxiously the arrival of the first load of patients, having been already notified of the commencement of the battle by the thunder of the artillery. Here begins the accurate examination of patients, who were only temporarily dressed on the field, and here operating goes on most actively. Amputations, resections, extractions of bullets, etc., are done by wholesale, and the surgeon who has been anxious for his first operation on a living patient may operate till he stops from exhaustion. This continues till far into the night; the fight lasts till late in the evening, and it is near morning before the last loads of wounded come in. With bad lights, on a temporary operating-table, and often with unskilful nurses for assistants, the surgeon must at once examine every patient, down to the last, and then operate and dress his wounds. In the field-hospitals the wounded have a period of rest, and, if possible, those who have been operated on or are seriously hurt should not be moved to another hospital till healthy suppuration begins and healing has at least commenced. This cannot always be done. Occasionally the place where the field-hospital has been established must be vacated. If one belongs to the vanquished party, and the enemy takes the place where the field-hospital was established, the surgeons are usually taken prisoners with their wounded; for, even when the enemy is most humane, after a great battle there is often such a demand for surgeons that those of the enemy cannot take the proper care of wounded prisoners. A few years since, in Geneva, a convention of European powers determined that surgeons and sanitary supplies should be considered neutral. Although there are some practical difficulties in carrying out this principle, it has done great good in the wars of late years, and is capable of still further development. At all events, the idea of considering a wounded enemy as an enemy no longer, but as a patient, is to be prized as a beautiful evidence of advancing humanity.

When the wounded have all been brought under cover, bedded, and the necessary operations done, and the diet, etc., has been attended to, arrangements should be made for their proper disposition. Permanent collection of many wounded men in one place is injurious, and, when the seat of war is a poor country, with few railroad connections, the care of the wounded is particularly difficult. Hence, they should be sent off as soon as possible. This may be done, even with the severely wounded, when there is a railroad handy; when the transportation is less convenient, the more slightly wounded at least can be removed. This system of scattering, which of late has been conducted with excellent results, requires great circumspection and trouble from the superior medical and military authorities, but it has proved advantageous. If houses (barracks), or, in summer, tents, can be erected for those remaining—the severely wounded—that will be best. If this be not practicable, they may be distributed in private houses; it has proved unadvisable to leave the wounded in school-houses and churches.

The war in North America, as well as that between Austria and Prussia in 1866, showed that there were still improvements to be made in military sanitary arrangements. A factor has been added that never before came as an aid, namely, extensive assistance from societies, Sisters of Charity, civil surgeons, and many other persons who, either personally or by money and stores, aided in the care of the wounded. When this private aid is properly organized, under proper management of the military officers, it may be very useful.

Concerning the treatment of gunshot-wounds, views have greatly changed from time to time, according to the point of view from which they were regarded. The oldest surgeons whose opinions we have, considered them as poisoned, and thought, consequently, that they should be treated with the hot iron or boiling oil. The first to oppose this view successfully was *Ambrose Paré*, whom you already know to have introduced the ligature for arteries. He relates that in the campaign in Piedmont (1536) he ran short of oil for burning the wounds, and he expected the death of all the patients who could not be treated according to the rules of the time. But this did not happen; on the contrary, they did better than the chosen few on whom he used the remains of his oil. Thus a lucky accident tolerably soon freed medicine of this superstition. Later it was very correctly observed that the great difficulty in healing gunshot-wounds was due to the narrowness of the canal, and attempts were made to obviate this by plugging the wound with charpie or gentian-root. But sensible surgeons soon saw that this still more impeded the escape of pus from the deeper parts, and the correct view commenced to make some headway, that a gunshot-wound was a tubular contused wound. They sought to improve this in a peculiar way, by laying down the rule that every superficial gunshot-wound should be laid open, the opening of a canal leading into the deeper parts was to be enlarged by one or more incisions; various methods were proposed for changing the contused wound into a simple incised wound by these incisions, while, in fact, the only thing that was done was to add an incised wound to the gunshot-wound. The case was somewhat different when the rule was given to cut out the whole course of the canal, and close the resulting canal by sutures and compresses, so as to obtain healing by first intention; this proceeding cannot often be applied, and obtained little reputation. Of late, since the treatment of all wounds is so much simplified, the same thing has happened to gunshot-wounds which are treated on the same general principles as contused wounds. In these, as in other wounds, the first thing is to arrest any arterial hæmorrhage. This is to be done according to the rules already given, the bleeding artery being tied either in the wound itself, or the corresponding arterial trunk being ligated in its continuity; to accomplish the former, it is generally necessary to enlarge the opening of entrance or exit, otherwise we should not find the bleeding artery. If there be no hæmorrhage, we should examine the wound, especially any blind canal, for foreign bodies, particularly for the bullet. This may be done most certainly with the finger; should it not be long enough, or should the canal be too narrow, we may best use a silver female catheter, with which we may feel more certainly and safely than with a probe; if we feel the bullet, we try to remove it the shortest way, that is, either draw it out at the point of entrance, or, if it lies in a blind canal, close under the skin, we make an incision through the skin and extract it through this, thereby changing the blind canal into a complete one. The extraction of bullets through the opening of entrance may be made by aid of spoon or forceps-shaped instruments. Bullet-forceps with long, thin blades are often difficult to use, because they cannot be sufficiently opened in the narrow canal to seize the bullet, hence many military surgeons prefer the spoon-shaped instrument. Such a bullet scoop has lately been suggested by *B. v. Langenbeck*, and seems very practical; in it the spoon is movable so as to pass behind the bullet, and push it forward. Still better, it seems to me, is a recently-invented American forceps, whose peculiarity is that they can be opened even in a narrow canal, and they seize very securely. If the bullet be lodged in a bone, we may bore a long gimlet into it,

Fig. 59.



Bullet-forceps, made by Geo. Tiemann & Co., of New York, with sharp points for seizing leaden bullets.

and try to extract it in that way. If we do not succeed in removing the bullet or other foreign body by the opening of entrance, we proceed to enlarge it to gain more room so as to apply the instruments better. The experience that bullets may often remain in the body without injury should warn us against any violent operation that aims only at their extraction. Hence, hæmorrhage and difficult extraction of foreign bodies are the chief indications for primary dilatation of gunshot-wounds. Later, other indications may arise to necessitate it; but, in the gunshot-wound, such enlargement is not necessary for a cure. This takes place by the throwing off of a small ring-shaped eschar, and the detachment of gangrenous shreds from the track of the wound, till healthy granulation and suppuration begin, and the canal gradually closes from within outward. In most cases the opening of exit cicatrizes before the entrance. Certain obstacles may stand in the way of this normal course; there may be deep progressive inflammations, rendering necessary new incisions and the employment of ice, as in other deep contused wounds.

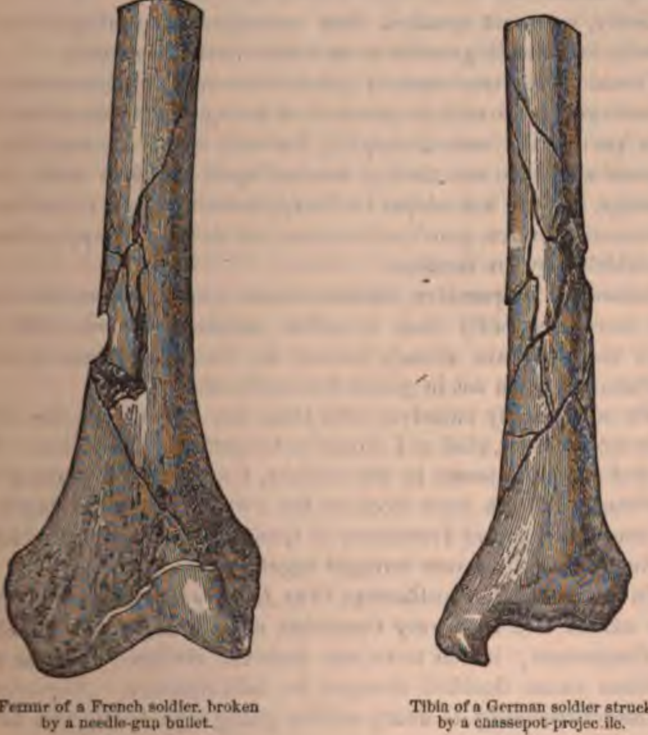
The first dressing of a gunshot-wound in the field is usually a moist compress, covered with a bit of oiled muslin or parchment-paper, held in place by a bandage or cloth. Frequently nothing further is required than simply keeping the wound moist and covered with charpie, lotions of lead-water, chlorine-water, etc. As yet there are no full observations of the treatment of gunshot-wounds without dressings. They occasionally, though rarely, heal by first intention; as a rule, they suppurate for a longer or shorter period. One of the chief causes of deep inflammation is the presence of foreign bodies, such as bits of clothing, leather, etc. The presence of the bullet, or a portion of it, is far less dangerous, for the cicatricial tissue may grow around and entirely encapsulate the lead, while the wound closes over it; the patient keeps the bullet in him. But these bullets do not always remain in the same spot; they partly sink, from their weight, partly are displaced, by muscular action, so that after years they are found at different (generally lower) points: for instance, a bullet may enter the thigh, and subsequently, after being almost forgotten, may be felt under the skin of the calf or heel, and may thence be readily extracted. I have told you the same thing about needles. But non-metallic bodies seem never able to remain thus without injury in the human body, and hence should always be extracted when discovered in a wound.

In gunshot-wounds the fever generally depends on their size and extent, as well as on the accidental suppuration. In the excellently-directed hospital of the Bavarian chief staff-surgeon *Beck*, which I visited at Tauberbischofsheim (1866), the thermometer was used for etermining the amount of fever; the results as to fever generally orrespond with those in other injuries.

[*Demarquay* (quoted in the *Medical Times and Gazette*, September, 1871) says that in all cases observed, where the temperature fell below 95° Fahr., the patients died.]

The special rules to be observed in perforating wounds of the skull, thorax, and abdomen, are given in special surgery; let us here make a few remarks on the fractures resulting from gunshot-wounds. We have already stated that simple subcutaneous fractures occur from spent or obliquely-falling bullets; but, in most cases, the fractures are accompanied by wounds of the soft parts. The soft, spongy bones and the epiphyses may be simply perforated by bullets without any splintering. This injury is comparatively favorable; if the adjacent joint be not opened, the bullet may remain in the bone, and, if it cannot be extracted, may heal there; the track of the wound in the bone suppurates, fills with granulations, which at least partly ossify, so that the firmness of the bone is not impaired. If the bullet strikes

FIG. 60.



Femur of a French soldier, broken by a needle-gun bullet.

Tibia of a German soldier struck by a chassepot-projectile.

the diaphysis of a long bone, it generally splinters it, and does so much more extensively than any other cause. The numbers of sharp splinters, and the extent of the splintering in proportion to the diameter of the projectile, is the most noticeable feature that we observe when first seeing a large number of gunshot-wounds.

I think it is necessary and very important to examine every gunshot-wound of the extremities with the finger quite early, and to remove fragments that are loose or slightly attached to the soft parts; it may be advisable now and then to cut or saw off pointed fragments where it can be done without much new injury or extensive incisions through the soft parts. But I would not recommend these *resections in the continuity* as a usual or necessary operation, for experience shows that many such cases go on favorably without operations.

If the injury has caused a complicated fracture in a joint, we cannot hope for much from an expectant treatment, according to present experience, which is based on statistics; the question rather seems to be, whether primary resection or amputation is preferable; this can only be decided by the peculiarities of each case.

Lastly, we must mention that secondary hæmorrhages are particularly frequent in gunshot as in other contused wounds.

I consider the treatment of gunshot-fractures, by fenestrated plaster-bandages, as the only proper method (excepting perhaps those in the upper part of the arm or thigh); the only thing against it is, that surgeons who have not already treated open fractures with plaster-dressings, and are not adepts in the application, should not make their first experiments on gunshot-fractures, but should only apply dressings with which they are familiar.

Secondary suppurative inflammations occur in gunshot-wounds even more frequently than in other contused wounds; the same causes that we have already learned for these dangerous accidents, unfortunately often act in gunshot-wounds also.

We must satisfy ourselves with these few remarks on the subject of gunshot-wounds, glad as I should be to continue the subject. Those who feel special interest in the subject, I refer to the works already mentioned, and to a little book of my own, "Historical Studies on the Consideration and Treatment of Gunshot-Wounds," in which you will find the old literature brought together.

[In the recent Turko-Russian War *Lister's* treatment was tried to some extent and with very favorable results, as shown by *Reyher* and *Bergmann;* in the next war between civilized nations it will doubtless cause decided changes in field-practice. *Esmarch* has proposed furnishing to every soldier going into battle two balls of salicylated wadding done up in gutta-percha cloth, with which to cover a gunshot-wound either in himself or a comrade; the wadding unites with the blood to form a provisional antiseptic dressing till the patient comes under the surgeon's care. *Esmarch's* experience shows that this dressing may stay on several days, and that healing under it may occur without reaction: although healing by first intention has been observed, it is very rare; gunshot-wounds usually suppurate, but under *Lister's* dressing small wounds, as those made by pistol-balls, not unfrequently heal by second intention without suppuration. *Klebs* made some very accurate pathologico-anatomical observations on gunshot-wounds in the late German-French War; he saw perforating wounds of chest and abdomen without suppuration or inflammation of their walls. *Klebs* supposed that in these cases valvular closure of the skin-wound prevented the entrance of air, which explained the absence of irritation from the injury; such results are rare, and are not to be expected.

Antiseptic treatment of gunshot fractures is entirely analogous to that of other compound fractures. Much depends on the injury to the soft parts; if they are much crushed, permanent irrigation may be indicated; otherwise the seat of fracture may be left exposed, disinfected, washed with concentrated solution of chloride of zinc, and placed in a *Lister's* dressing and in splints, or a fixed dressing. If the wound be not too long or deep, it will be best to lay it open, remove the crushed tissue, disinfect, drain, and then unite by deep and superficial sutures; this gives better chance of healing without reaction than when we have to disinfect by injections, etc.

At present with *Lister's* dressing we apply wooden or gutta-percha splints, or else permanent extension instead of fenestrated plaster splints, as formerly.]

# CHAPTER IX.

## *BURNS AND FROST-BITES.*

### LECTURE XX.

1 **Burns:** Grade, Extent, Treatment.—Sunstroke.—Stroke of Lightning.—2. **Frost-bites:** Grade.—General Freezing, Treatment.—Chilblains.

THE symptoms due to burns and frost-bites are quite similar, but are sufficiently distinct to be regarded separately; we shall first treat of

BURNS.

These are caused by the flames, when, for instance, the clothes burn, but more frequently by hot fluids, as when children pull vessels of hot water, coffee, soup, etc., off a table on to themselves. And, unfortunately, in factories, burns from hot metals, such as molten lead, iron, etc., are not rare, and in every-day life slighter burns from matches, sealing-wax, etc., often occur, as you have all doubtless seen. Besides the above, concentrated acids and caustic alkalies not unfrequently cause burns of various degrees, analogous to those from hot bodies.

In burns the intensity and extent of the injury are to be regarded; we shall hereafter study the latter. The *intensity* of the burn depends essentially on the grade of the heat and the duration of its action; according to the result of this action, burns have been divided into three grades. These pass into one another, but from the accompanying symptoms may be distinguished without difficulty; the only object of this is to render explanation easier. We assume three grades.

*First degree* (hyperæmia): The skin is much reddened, very painful, and slightly swollen. These symptoms are due to dilatation of the capillaries, and slight exudation of serum in the tissue of the cutis. There is a mild grade of inflammation, in which there is an increase of cells in the rete Malpighii alone, which is followed, in many cases at least, by detachment of the epidermis. Redness and pain occasionally last a few hours, in other cases several days. But it is not necessary, and not at all practical, to make several grades on this account.

*Second degree* (formation of vesicles): Besides the symptoms of the first degree, vesicles arise on the surface of the skin; before bursting these contain serum, clear or mixed with a little blood. These vesicles form immediately, or in a few hours after the reception of the burn, and may vary greatly in size. Anatomically we find that in most of these cases the horny layer is detached from the mucous layer of the epidermis, so that the fluid rapidly escaping from the capillaries lies between these two layers, just as results from the action of a blister. The vesicles rupture or are punctured; from the remaining rete Malpighii a new horny layer of the epidermis forms quickly, and in six or eight days the skin is the same as before. It may also happen that after removal of the vesicle the denuded portion of skin is excessively painful, and for several days, or even a fortnight, there may be superficial suppuration; the pus finally dries to a scab, under which the new epidermis forms. You may induce this state also artificially by leaving a blister for a long time on one spot. Here also it is unnecessary to make new grades of these variations, for they only depend on a little greater or less destruction of the rete Malpighii, while the greater or less pain corresponds to the amount of denudation of the nerves in the papillæ of the skin.

*Third degree* (formation of eschars): By this term we may designate all those cases where there is formation of eschars, i. e., where portions of the skin, and even of the deeper soft parts, are destroyed by the burn. Of course, the varieties may be very great, as in one case there may be only burning and charring of the epidermis and papillæ, in another death of a portion of the cutis, in a third charring of the skin or of an entire limb. In all cases where the papillary layer, with the rete Malpighii, is destroyed, there will be more or less suppuration, by which the mortified portion will be detached, which of course will leave a granulating wound, that will follow the ordinary course in healing. If only the epidermis and the surface of the papillæ be charred, there is only slight suppuration, with rapid replacement of the epidermic layer from the remains of the rete Malpighii.

From what has been said, you may understand how from four to seven or more degrees might be formed; but, to make the subject comprehensible, the three degrees of redness, vesicles, and eschars, are enough. In extensive burns we often find these different degrees combined, and, when the injured part is covered with charred epidermis and dirt, it is often difficult to determine the degree at any point. If there be suppuration, it may be either superficial or deep; occasionally it appears as if islands of young cicatricial tissue formed in the midst of a granulating wound, and this has given rise to the false idea that the latter could cicatrize not only from the edges but from different points in the midst of the wound. But such cicatricial islands never form where there is total absence of the papillary bodies of the skin, but only from some remnants of the rete Malpighii, as may happen in burns and certain ulcerations to be hereafter mentioned.

The prognosis for the function of burnt parts may be inferred from what has been said. We should, however, add that after extensive loss of the skin, as occurs especially from burns of the neck and upper extremities by hot liquids, there is very considerable cicatricial contraction, by which, for instance, the head may be completely drawn to one side of the neck, or anteriorly to the sternum, or the arm fixed in a flexed position by a cicatrix in the bend of the elbow. In the course of time these cicatrices become more distensible and pliable, but rarely to such an extent as entirely to remove the disturbance of function and the disfigurement, so that in many cases plastic operations are necessary to improve these conditions. It was formerly asserted that the cicatrices after burns contracted more strongly than any other cicatrices. But this is only apparently so, for scarcely any other injury ever causes the loss of such large portions of skin; we may readily perceive that, when this does occur (as in plastic operations and after extensive destruction of the skin by ulcerations), the contraction of the cicatrix is just as great.

Entirely apart from the different degrees of burns, their *extent* is of the greatest importance, as regards their danger to life. It is generally said that, if two-thirds of the surface of the body be burned only in the first degree, death soon occurs, in a manner that has as yet received no physiological explanation.

[When granulating wounds began to skin over from the middle, it was supposed that the epithelium of the cicatricial islands was formed from granulations or wandering cells, and that an epithelial matrix was not necessary for cicatrization; but numerous accurate observations seem to show that this view is untenable, and that in such cases the burn has been of unequal depth. The papillary layer of the skin has been destroyed, but at some points the sweat-glands and hair-follicles have remained intact; if the entire surface of the wound now granulates, the epithelial remnants are at first overgrown and escape notice, but, as the granulations change to connective tissue, they start up as islands between the granulations, and cicatrization proceeds from them as from the edges.]

Should the burn not prove fatal from its extent alone, the great loss of skin and consequent suppuration may prove dangerous, especially for children and old persons; in the same way the amputations necessary from complete charring of single extremities involve certain dangers, which are the more serious as they affect persons already greatly depressed by the burn.

In the *treatment* of burns in the first and second degrees, more depends on alleviating the pain than on any energetic treatment; for we cannot hasten the return of the skin to its natural state, but must leave the course of healing entirely to Nature.

[Immediately after an extensive burn, even when it has not been deep, the patient is usually very much excited, crying and moaning with pain, but when the pain is alleviated he quiets down, and then only wants to drink. Consciousness is perfect—the most exact account of the injury can be given. In children particularly, very soon after severe burns, there is vomiting of food and bile, rarely of blood; the latter is almost always a sign of fatal result. The patient does not pass water, and if a catheter be introduced we find no urine, or only a small quantity of albuminous or hæmorrhagic urine. A few hours after the accident there is yawning and deep sighing; the patient gradually becomes apathetic; if he has not already vomited, he has eructation and hiccough, and perhaps vomiting. Then delirium begins, the patient tosses about regardless of his burns, he has clonic spasms, sometimes opisthotonus; he becomes unconscious; the pulse, which just after the injury was frequent and small, becomes threadlike and cannot be counted, the respiration is hurried and superficial, there is cyanosis, and the patient dies in delirium or stupor a few hours or one or two days after the injury. Very early the bodily temperature falls below normal, and remains so till death; this low temperature is of bad prognosis. In other cases life lasts longer; the patient appears to revive, but, even after a week, the above symptoms may come on and cause death very suddenly.

The fatal result may be due to intense phlogistic or septic poisoning, especially in rather chronic cases.

*Wertheim* first called attention to the presence of peculiar bodies in the blood of patients who had been burned, which he recognized as destroyed red blood-corpuscles; hence he concluded there was extensive destruction of the blood; but this view has been contradicted by others. *Ponfick* has shown by experiments on dogs that even in a few minutes after reception of a burn there is a marked change in the blood, a molecular disintegration of the red corpuscles. The hæmoglobin thus set free is excreted by the kidneys and excites severe parenchymatous nephritis; so the actual cause of death is destruction of the red corpuscles and liberation of hæmoglobin. *Sonnenburg's* experiments show that death coming very quickly after burns is due to overheating of the blood and consequent paralysis of the heart. If death does not occur at once, the blood-pressure sinks from reflex paralysis of the vessels, which, however, does not take place if the spinal medulla has been divided. In such cases, burns are much better borne; from these results *Sonnenburg* thinks that reduced tone of the vessels induced by abnormal irritation of the nervous system is the actual cause of death in extensive burns. Another view, held by *Hebra*, among others, is that death after burns is due to shock, that is, to the effect on the sensitive nerves, and consequent reflex action on the heart. We know of no remedy for avoiding the impending death from severe burns. *Ponfick*, on theoretical grounds, recommended transfusion of healthy blood after removal of a corresponding quantity of the changed blood; but at present we have not enough evidence as to the effect to come to any decision. [*Ponfick*, as quoted in the "Medical Record" from the "Med. Chir. Rundschau," 1879, and the "Allg. Med. Cent. Zeitung," No. 4, 1880, transfuses by injecting defibrinated blood into the peritoneal sac. It is stated that this is followed by very little febrile reaction; and that after the operation the proportion of red corpuscles in the blood of the patient operated on is at once increased. *Ponfick* tried the operations on animals, then on human patients; but the translator does not know of its having been done by any one in America.] In burns of the first and second class our first care should be to relieve pain; this is generally done by covering the burned skin, so as to protect the exposed ends of the nerves from the air.

If the burn is very extensive, cold applications might reduce the temperature so as to hasten collapse; even when not extensive, it is not always well borne. In burns of the first class we may simply sprinkle on starch, flowers of zinc, bicarbonate of soda, smear with oil, or apply mashed potato, starch, etc.; the whole burn may be enveloped in prepared or salicylated cotton moderately compressed by bandages; this may be left on till thrown off by the wound, and new layers applied over it with fresh bandages.

In every case, but particularly where the burn is deep, accurate antiseptic treatment should be employed whenever possible; not only because we thus avoid frequent change of dressings, which is very painful, but elimination of the gangrenous parts goes on without much general or local reaction; the slough may even be detached without inflammation, and healing go on without suppuration, if the burn has not been too extensive.]

Another plan of treatment is with a solution of nitrate of silver, ten grains to the ounce of water; this is to be painted over the burnt part, and compresses wet with the same to be kept constantly applied. At first the pain from the cauterization of the parts denuded of epidermis is occasionally very great, but a thin blackish-brown crust soon forms, and the pain then ceases entirely. I particularly recommend to you this plan of treatment when all three degrees of burns are combined.

In burns of the third degree, if there is only mortification of the cutis (when this is not charred, but burned by boiling water, it generally becomes perfectly white), the treatment is the same as that above given. Should it subsequently be desirable to hasten the detachment of the eschar, cataplasms may be employed to stimulate suppuration; in most cases, however, this will be unnecessary, and the treatment by nitrate of silver may be continued till the eschar is completely detached. If large granulating surfaces remain, especially on parts of the surface that are moved much, and where the neighboring skin is not very movable, it may take a long time, often months, for them to heal. Very luxuriant granulations form, and their tendency to cicatrize is always very slight. Of the remedies already given for promoting the healing of such wounds, I particularly recommend to you the compression of the wound by strips of adhesive plaster, which are of excellent service in some of these cases. In the treatment of cicatricial contractions resulting from these burns, compression of the cicatricial bands by adhesive plaster is one of the most important remedies, and you would always do well to try this persistently before resorting to excision of the cicatrix, or to plastic operations.

If, in a burn of the third degree, there has been charring of a limb, it may often be advisable to amputate at once; not only because the detachment of a large part of the body is not free from danger, but also because the stumps thus left are unfit for the application of an artificial limb.

If called to a case where there is a burn of the greater part of the body, you must give your whole attention to the general condition of the patient, and try to prevent collapse, by the use of stimulants, such as wine, hot drinks, hot baths, ether, ammonia, etc. Unfortunately, in most of these cases, our efforts to preserve life are in vain. *Hebra* praises the treatment of extensive burns by the continued warm bath, which, under proper circumstances, may be kept up for weeks.

[*Hebra's* water-bed, elsewhere spoken of, is a bath-tub containing a frame suspended by chains; the frame has a covering on which the patient lies, and by means of a windlass he can be raised out of the water and lowered again; his head rests on a cushion. The bath is filled with water at a temperature of 86°, and the patient put in without any dressings; soon the temperature must be raised to 101° or 104°, else he will suffer from cold. The water is to be kept at this temperature, which gives the patient great comfort; pain ceases, excitement is allayed. The patient remains in the water-bed day and night; when he wishes to pass water or have a movement from the bowels, he is raised on the frame. From severe burns patients die in the bath as often as under other treatment, but they do not suffer so much; and if the burn be not so extensive as to induce death quickly, the bath is an excellent means of treatment, and saves the patient much pain. He may, without great annoyance, stay in the bath for weeks or months. The water protects the parts from the air, prevents decomposition of the slough and favors its detachment, renders bandages and dressing unnecessary, limits suppuration, and hastens cicatrization. Unfortunately, *Hebra's* water-bed, which is also useful in extensive gangrenous wounds and ulcers, and in many skin-diseases, is only found in hospitals; hence, in private practice we must rely on other modes of treatment, and especially on antiseptic dressing.]

Persons with delicate skins, long exposed to the *sun's rays*, may have slight degrees of burns of the face and neck. This is often observed in persons travelling on the mountains. When persons, especially women, who do not usually pass the day in the sun, travel for several bright days in summer, without carefully protecting the face and neck, the skin becomes red, swollen, and very painful; after three or four days the skin dries to brown crusts, cracks, and peels off. In other persons, with still more irritable skins, vesicles form, which subsequently dry up, without, however, leaving any cicatrices (eczema solare). Besides prophylaxis by veils, sun-shades, etc., it is well to cover the skin of such mountain travellers with cold cream or glycerine; the same remedies may also be used in developed sunburn; if the burnt parts be very painful, we may apply cold compresses.

Here we must also speak of *sunstroke*, or *insolation*. In our climate, this disease occurs almost exclusively in young soldiers, who have to make fatiguing marches in full uniform in very hot, bright weather. There are severe headache, dizziness, unconsciousness, and sometimes death in a few hours. In the Orient, especially in India, this disease is not rare among the English soldiers; some cases are quite acute, ending with tetanic spasms; others begin with long prodromata, and drag on with symptoms of severe headache, burning skin, continued fatigue and depression, palpitation of the heart, twitching of the muscles, etc.; even when this state ends in recovery, relapses are common. Patients with sunstroke are to be treated like those with congestion of the brain. Cold affusions and bladders of ice to the head, rest in a cool chamber, purgatives, leeches behind the ears, sinapisms to the nape of the neck, are the proper remedies. According to the experience of English surgeons, venesection is injurious.

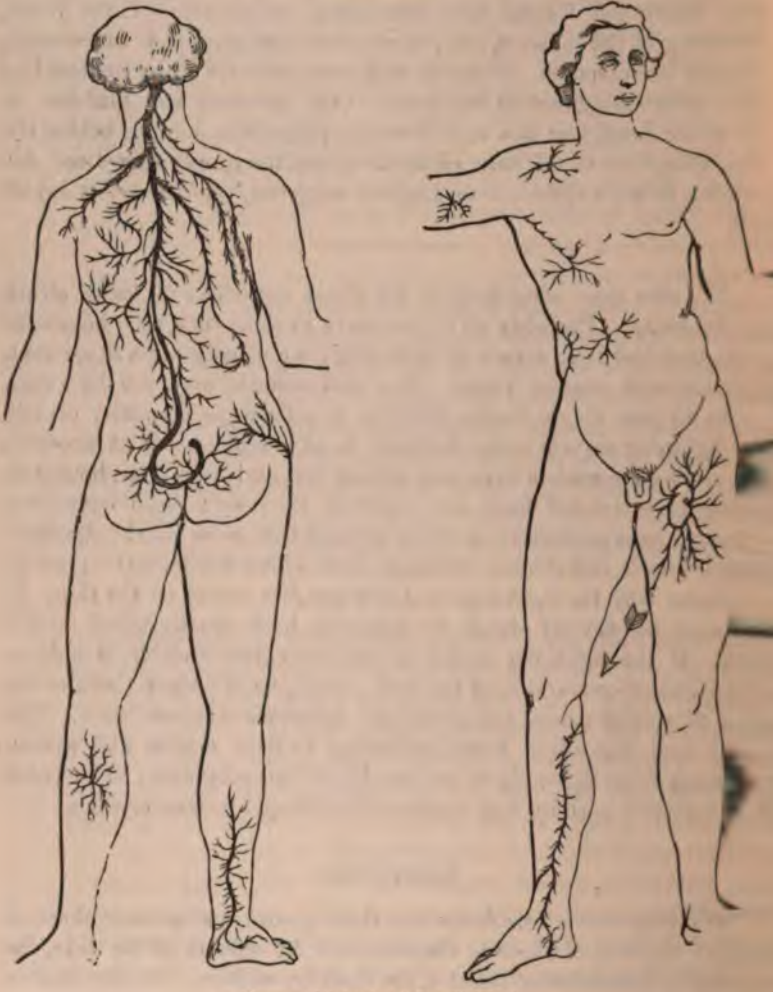
* * *

We also have something to say about the effect of being struck by lightning. Probably all of you have at some time seen houses or trees that had been struck by lightning; we usually see a large rent, a fissure with charred edges. Men and animals may also be struck so as to lose single limbs, but this is not always the case; usually the lightning travels along the body, in at one place, out at another; the clothes are rent, or even torn off and cast aside; peculiar, branched, zigzag brownish-red lines are found on the body; these have been regarded as representations of the nearest tree, or as blood coagulated in the vessels and shining through; both views are incorrect; we do not know why the lightning runs this peculiar course on the skin. If a person be directly struck by lightning, he is usually killed on the spot. If the lightning strike in his immediate vicinity, it induces symptoms of commotion of the brain, paralysis of certain limbs or organs of special sense, and occasional extravasations and burns. The latter heal like other burns, according to their degree and extent. Paralysis from lightning is not usually of bad prognosis; the nervous and muscular activity may return after a longer or shorter time.

## FROST-BITES.

We may divide frost-bites into three grades analogous to those of burns; the first of these is characterized by redness of the skin, the second by formation of vesicles, the third by eschars. The first degree of frost-bite is quite well known; we might regard the so-called deadness of the fingers as its mildest form; probably each of you has sometime had this in a cold bath, or in winter-time. The finger becomes white, the skin wrinkled, the sensation diminished; after a time these symptoms pass off, the skin becomes red, the finger swells, and there is a peculiar itching and prickling. This increases the more, the more quickly warmth follows the cold. The redness of the skin of this degree of frost-bite differs from that in burns, by its more bluish-violet color.

FIG. 61.



Traces of lightning (after *Stricker*).

After a time, these symptoms subside and the skin again becomes normal. Generally no remedies are used in these slight cases, but, very properly, patients are warned against warming the parts too rapidly; rubbing with snow, then gradually elevating the temperature, is recommended. The above symptoms are thus explained: First, the capillaries are strongly contracted by the cold, and are then paralyzed for a time. I shall not here discuss the tenability of this hypothesis; this explanation involves all the difficulties that we have already met in the theories of inflammation.

[In some persons the least cold causes contraction, not of the capillaries, but of the arteries, especially in the fingers, usually only in one or two fingers. Dipping the hand in cold water, even in summer, or a damp atmosphere, is enough in some persons to induce actual contraction of the artery; the finger looks like that of a corpse; it is absolutely anæmic, as if it had an *Esmarch's* bandage applied; pricking with a needle brings no blood; at the same time there is a feeling of numbness and stiffness. Restoration of the circulation takes ten or fifteen minutes; rubbing, warming, etc., have little effect; only after dipping in *hot* water does the circulation gradually return; the first phalanx reddens while the others remain white for a time. There is no pain, and very little paralytic dilatation of the vessels. The whole process differs entirely from ordinary contraction of the capillaries induced by anæmia from cold, for only a certain vascular territory, which is always the same, is affected.

In cases of freezing, if there is formation of vesicles, there is more danger than if there is merely redness; they are often accompanied by loss of sensation, and there is danger of mortification. Freezing differs from burning; in the latter, gangrene is an immediate result; it does not extend subsequently; but in freezing, the cold acts first on the tissues and the fluid contained in them, and it is only when the parts have thawed that the changes leading to mortification show themselves; so, at first, the parts may look normal, and there may be some circulation, while the vitality of the cells and blood has been so much affected that gangrene is inevitable; but we cannot say how far it will extend.

Blood that has been frozen and thawed out is bright red, but the hæmoglobin escapes from the cells; this blood has the peculiarity of causing coagulation in normal blood, and by injecting enough of it into the circulation of a living animal you cause its death from clots in the heart and large blood-vessels.]

Recent investigations of *Samuel* show that after certain grades of freezing there is a true inflammation which goes on to regular gangrene. From clinical experience I know that in such cases there is a process not found in burns; for parts badly burned, even when not turned to cinder, shrivel up, and the blood coagulates in the vessels, so that other blood cannot enter them even if they continue to exist. If a frozen limb thaws for a time, arterial blood may again enter the vessels, and the question will be whether the walls of the vessels can still keep the blood fluid and the tissues use up the blood coming to them. If this be so, the frozen limb may regain its vitality; if it does not, gangrene occurs. In this transition stage the veins are much distended, and this may facilitate thrombosis in them. *Bergmann* recommends particular attention in the treatment of this stage; he has had unusually good results from vertical suspension of the limb, which favors the return of the venous blood.

Redness following a frost-bite may sometimes remain permanent, i. e., the capillaries remain dilated. This is especially apt to occur in frost-bites of the nose and ears, and is usually incurable. In Berlin, I treated a young man who had a dark-blue nose, as a result of frost-bite, and wished at all hazards to be relieved of the deformity. He persistently pursued the different modes of treatment; first, he had the nose painted with collodion, after which it looked as if varnished, and, as long as the coating of collodion continued, it was somewhat paler, but the improvement was not permanent. Then the nose was painted with dilute nitric acid, which gave it a yellow tint. After detachment of the epidermis the evil again appeared improved for a time; but it soon returned to its former state. Then we tried treatment with tincture of iodine and nitrate of silver, which for a time gave the nose a brownish-red, then a brownish-black color. The patient bore all these changes of color heroically, but the perverse capillaries continued dilated, and the nose remained bluish red at the last, just as it had been. I still thought of trying cold, but feared the condition might be made worse, and, after several months' treatment, had to tell the hero of this tragi-comical history that I could not cure him. The treatment of chilblains and the consequent ulcers, of which we shall speak immediately, may be just as difficult.

Frost-bite, where, besides redness of the skin, there is formation of vesicles, is more severe; it is often accompanied by complete loss of sensation of the affected part, and there is always danger of mortification. The formation of vesicles in frost-bite is prognostically much worse than it is in burns. The serum contained in the vesicles is rarely clear, but usually bloody. A limb completely frozen is said to be perfectly stiff and brittle, and small portions are said to break off like glass, if carelessly handled. I have had no opportunity to verify these statements, but remember that, when I was a student, a man was brought to the Göttingen surgical clinic with both feet frozen; during transportation to the hospital, they had become spontaneously detached at the ankle-joint, so that they hung only by a couple o tendons. Double amputation of the leg above the malleoli had to made. How far a limb may be entirely frozen, so that the circulati is entirely arrested, frequently cannot be determined for a time; hence we must not be too hasty about amputating. In Zürich, I had two cases where both feet were dark blue and without feeling, and on being punctured with a needle only a drop of black blood escaped; nevertheless, the foot lived, and only a few toes were lost. In a third case, in a very debilitated patient, where both feet as high as the calf were dark blue and covered with vesicles, they became entirely gangrenous. If there be extensive gangrene of the skin, beyond a doubt, we should not delay amputating, for these patients are very subject to pyæmia. A very sad case occurred in the Zürich hospital. A powerful young man had both hands and both feet frozen, so that all became gangrenous; the patient could not make up his mind to the four amputations, nor could I bring myself to persuade him to the fearful operation. He died of pyæmia.

The ends of the extremities, the point of the nose, and tips of the ears, are most liable to be frozen. Closely-fitting clothes, which impede the circulation, increase the predisposition. Cold wind, and cold accompanied by moisture, induce frost-bite more readily than very great still, dry cold.

There is also a total freezing or stiffening of the whole body, in which the patient loses consciousness, and falls into a state of very limited vitality. The radial pulse can hardly be felt, the heart-beat is scarcely audible, the respiration almost imperceptible, and the whole body is icy cold. This state may pass at once into death; then all the fluids harden into ice. This general freezing is especially apt to occur when the individual, overcome by fatigue and cold, lies down while freezing; he soon falls asleep, and sometimes never wakes again. It has never been accurately determined how long a patient may remain in this stiff condition, with very slight appearance of life, and again recover; we find mention of the state having lasted six days. Whether this be true or not, we should continue our attempts at resuscitation as long as a heart-beat can be detected.

Let us commence the *treatment* of frost-bite with this state of general stiffness. We must here avoid any sudden change to higher temperature, but increase the warmth gradually. Such a patient should be placed in a cool chamber, on a cold bed, and frictions made for several hours. At the same time, artificial respiration should be occasionally tried, if the breathing becomes imperceptible. As slight stimulants that may do good, I would mention enemata of cold water, and holding ammonia to the nostrils. Very gradually, as the patient becomes conscious, we raise the surrounding temperature, keep him for a time in a slightly-warmed room, and at first give only tepid drinks. As the different parts of the body, one by one, regain vitality, there is occasionally some pain in the limbs, especially if they were warmed too rapidly; in these cases it is well to envelop the painful parts in cloths dipped in cold water. The patient may remain for hours or days in a benumbed, senseless condition, which disappears gradually Of late, experiments have been made in resuscitating stiffened animals, which appear to show that animals are more certainly saved from death by rapid than by slow warming. I should not readily decide, from these experiments on animals, to depart from the rules already empirically employed for treatment of persons frozen stiff, and which appear to be correct for local frost-bites, but the question is worth further experiment. Such cases of general freezing rarely escape without loss of some limbs, or parts of them, and, in regard to the treatment of these frozen parts, I can give you little advice. The vesicles are punctured and evacuated; the feet or hands may be wrapped in cold, wet cloths; then we must wait to see whether and how extensively gangrene will occur. If the bluish-red color passes into a dark cherry-red, the chances of restoration to life are slight. Gangrene will occur in the great majority of such cases. By testing the sensibility with a needle, and noting the escape of blood from these fine openings, we test how far the limb has ceased to live; but this only becomes certain when the line of demarkation forms; that is, when the dead is sharply bounded from the living, and inflammatory redness develops on the border of the gangrenous parts. But the general condition may become dangerous before the line of demarkation is fully formed; hence amputation must not be delayed too long if the inflammation after freezing assumes a phlegmonous character. The detachment of single toes or fingers we may leave to itself; but where there is gangrene of a large part of a limb, amputation is decidedly preferable.

I will here return to chilblains (perniones), not because they may become particularly dangerous, but because they are an exceedingly annoying disease, and are in some cases very difficult to cure, and for which, as good family doctors, you must have a series of remedies. Chilblains are caused by paralysis of the capillaries, with serous exudation in the tissue of the cutis; they are, as most of you know, bluish-red swellings on the hands and feet, which prove excessively annoying from their severe burning and itching, and from the occasional formation of ulcers. They result from repeated slight freezing of the same spot, and do not occur with equal frequency in all persons; they are less annoying in very cold weather than during the change from cold to warm. At night, on going to bed, when the hands and feet become warm, the itching occasionally becomes so troublesome that the patient has to scratch them for hours. In general, females are more disposed than males, and young persons more than old, to chilblains. Employments requiring frequent change of temperature particularly predispose to them; clerks and apothecaries, who stay for a time in a warm room, then in a cold cellar or warehouse, are frequent subjects. But no station is exempt; people who always wear gloves, and rarely go out in winter, may be attacked as well as those who have never worn gloves. Among females, chlorosis and disturbances of menstruation occasionally seem to predispose to them; generally, frequent returns of frost-bite appear to be connected with some constitutional anomaly.

As regards treatment, it is usually very difficult to combat the causes due to constitution and occupation; hence we are chiefly limited to local remedies. In Italy, where the disease is very frequent, if a cold winter occurs, frictions with snow and ice compresses are recommended. With us, these are less used, and do no good, or at most only alleviate the itching for a time. Salve of white precipitate of mercury (one drachm to the ounce of lard), frictions with fresh lemon-juice, painting with nitric acid diluted with cinnamon-water (one drachm to four ounces), solution of nitrate of silver (ten grains to the ounce), and tincture of cantharides, are remedies that you may resort to. Sometimes one answers, sometimes another; hand or foot baths with muriatic acid (about one and a half to two ounces to a foot-bath, use for ten minutes), and washing with infusion of mustard-seed, are also celebrated. If the chilblains open on the top, they may be dressed with ointment of zinc or nitrate of silver (gr. j to ʒ j fat). I have here given you only a small number of the remedies recommended, the effect of most of which I have myself proved, although there are a number of others; at the commencement of your practice you will find these enough for combating this common, trifling disease.

# CHAPTER X.

## *ACUTE NON-TRAUMATIC INFLAMMATION OF THE SOFT PARTS.*

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## LECTURE XXI.

**General Etiology of Acute Inflammations.—Acute Inflammation: 1. Of the Cutis. *a*, Erysipelatous Inflammation; *b*, Furuncle; *c*, Carbuncle (anthrax), Pustula Maligna. 2. Of the Mucous Membranes. 3. Of the Cellular Tissue, Acute Abscesses. 4. Of the Muscles. 5. Of the Serous Membranes, Sheaths of the Tendons, and Subcutaneous Mucous Bursæ.**

GENTLEMEN: So far we have treated only of injuries, now we shall pass to the acute inflammations which are of non-traumatic origin. Of these cases, those belong to surgery that occur on the outer part of the body; also those which, occurring in internal organs, are still accessible to surgical treatment. Although I must start with the idea that you already know the causes of disease in general, it still seems necessary to make some preliminary remarks with special reference to the subject of which we are about to treat.

The causes of acute non-traumatic inflammations may be divided into about the following categories:

1. *Repeated Mechanical or Chemical Irritation.*—At the first glance, this seems to come under the head of trauma, but it makes considerable difference whether such an irritation acts once on a tissue or whether it be frequently repeated, for, in the latter case, each succeeding irritation affects a tissue already irritated. An example will make this clear to you. Suppose a person is rubbed continuously by a projecting sharp nail in his boot or shoe; at first there would be a slight wound with circumscribed inflammation, but afterward the inflammation will spread and become more intense as long as the irritation lasts. Let us take another example of chemical irritation: If a person not accustomed to highly-seasoned food eats Spanish pepper

at would induce temporary hyperæmia and swelling of the oral and gastric mucous membrane; should one continue the use of so acrid a substance for a length of time, he might excite a severe gastritis. Except in cases of the first example, these rapidly-repeated irritations are not frequent in practice, but they have a great deal to do with the origin of chronic inflammation; when, of themselves insignificant, they act on parts more or less weak. We must again return to this point.

2. *Catching Cold.*—You all know that by catching cold one may acquire various diseases, especially acute catarrh and inflammations of the joints or lungs; but we do not know what is the particular injurious influence in catching cold, or what immediate changes it causes in the tissues. The rapid change of temperature is blamed as the chief cause of catching cold, but by this means we cannot experimentally induce an inflammation, or any similar disease. One catches cold from being heated, and then being exposed to a cold draught for a length of time; by careful observation he may say just when he caught cold. The cold may have a purely local action; for instance, one sits for a time at the window, and the cold wind blows on the side of his face toward the window; after a few hours he is attacked by paralysis of the facial nerve. We may here assume that molecular changes have occurred in the nerve-substance, by which the conducting power of the nerve is lost. Another might get a conjunctivitis from the same cause. These are purely local colds. Another case is more frequent, viz., that on catching cold that part is attacked which in the person affected is most liable to disease, the "*locus minoris resistentiæ.*" Some persons, after catching cold in any way, have acute catarrh of the nose (snuffles); others have gastric catarrh, others muscular pains, and still others have inflammations of the joints. Now, as these parts are not always directly affected by the injury (as when one has nasal catarrh from getting his feet wet), we must suppose that the whole body is implicated, but the action of the injury is only shown at the *locus minoris resistentiæ.* Whether this transfer of such injurious influences to a special part of the body is due to the nerves, or to the blood and other fluids of the body, is a question which cannot at present be decided, and about which physicians are divided into the two great bodies of neuropaths and humoralists. Reasons may be adduced for both views. I rather incline to the humoral view, and regard it as possible that, for instance, chemical changes may occur or be prevented in the skin while sweating, which may have a poisonous effect on the blood, and may act as an irritant now on this, now on that organ. According to the old form of speech, these inflammations due to catching cold are called "rheumatic" (from ῥεῦμα,

flow); but this expression is so much misused, and has come into such disrepute, that it should not be employed too often.

3. *Toxic and Miasmatic Infection.*—We have already (page 169) stated that moist and dry, purulent and putrid, substances brought in contact with a wound induce severe progressive inflammations, if they enter the healthy tissue immediately after the injury or, under certain previously-mentioned circumstances, pass through the granulations of a wound into the tissue. It is true, the body is tolerably protected on its surface by the epidermis, on the mucous coats by thick epithelium, against the entrance of such poisonous and inflammatory materials, but the protection is not perfect. There are many poisonous substances which enter the body through the skin or mucous membrane. Some of them we term poison, such as the secretion from glander-ulcers in the horse, or from the carbunculous pustules in cattle; others we only know from their effects, from some circumstances of their origin. There are invisible bodies which we term "miasmatic poisons," or briefly "miasm" (*μιασμα*, uncleanness); it is supposed that these miasms develop from decomposing organic bodies. Some consider them as gases, others as dust-like particles, others as minute organisms or their germs; I think that in many cases the latter is the correct view. The action of these poisons varies, inasmuch as some of them have a direct phlogistic action; in others it is more indirect. Thus some poisons, as pus, cadaveric poison, induce severe inflammation at the point where they enter the body (*infectionsatrium*); others excite no inflammation at that point, but are imperceptibly taken into the blood, and, although circulating through all the organs, only have an inflammatory effect on one or a few parts of the body. These poisons are, to a certain extent, only injurious to certain organs; they have a "specific" action. I shall not here speak of the primary action of this poison in transforming the blood. We do not know the chemically active constituents of most of these poisons which act specifically on one organ or tissue; we cannot see them circulate, nor can we always see their effects. Hence, you may very justly ask me how we can express ourselves with so much certainty on the subject. We decide on the causes by observing the morbid symptoms, and, in so doing, support ourselves mainly on their analogy to the effects of poisons intentionally introduced into the body, especially to those of our most active medicines. If we take the group of narcotics, they all have a more or less benumbing effect, that is, a paralyzing effect, on the psychical functions, but they have also the most peculiar specific effects. Belladonna acts on the iris, digitalis on the heart, opium on the intestinal canal, etc. We see the same thing in other remedies. By repeated doses of ca

tharides we may excite inflammation of the kidneys, by mercury inflammation of the oral mucous membrane and salivary glands, etc., whether we introduce them into the blood through the stomach, rectum, or skin. So also there is an endless number of known and unknown organic septic poisons, of which many, if not all, have also a specific phlogogenous action. I mention only one example: if you inject putrid fluid into the blood of a dog, in many cases, besides the direct blood intoxication, he will have enteritis, pleuritis, or pericarditis. Must we not here suppose that the injected fluid contains one or more matters which have a specific inflammatory effect on the intestinal mucous membrane, on the pleura and pericardium? If we know the point of entrance of the poison, and have some experience of the poison itself, there will rarely be much doubt about the cause and action. But how many cases there may be where neither exists! I believe that infection is a much more frequent source of inflammations, especially in surgery, than has hitherto been suspected.

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I would still make a few general remarks about the *forms* and *course* of non-traumatic inflammations. I have already told you that the characteristic of traumatic inflammations is, that they are limited to the wounded part; if they become progressive, it is generally through mechanical or toxic (septic) irritation. This would imply that inflammations induced by mechanical irritations and toxic actions have a tendency to progress, or at least to diffuseness; this is true of most inflammations resulting from catching cold, which attack either a whole organ or a large section of one part of the body. In this regard, much depends on the intensity of the mechanical irritation, and, in toxic inflammations, on the quality and quantity of the poison, especially on its more or less intense fermenting action on the fluids permeating the tissues. As regards inflammations due to repeated mechanical irritation and catching cold, we do not always have reason to suppose that their products are more irritating than those of simple traumatic inflammation; but if, during the latter, the affected part be kept absolutely quiet, and the lymphatic vessels and interstices between the tissues are closed by the infiltration of the parts about the wound, the extension of the products of inflammation into the surrounding parts is much interfered with. But in repeated mechanical irritations the tissue is not kept at rest, and consequently the products of inflammation extend unimpeded around the irritated part, and excite new inflammation. In inflammation due to catching cold, according to my humoral view, the *materia peccans* is poured to a whole organ or tissue; hence, these inflammations are mostly diffuse from the commencement. If, from an existing point of inflammation, a phlogogenous material enter the blood, and thence specifically affect any other organ, we call this secondary inflammation "metastatic." But these metastatic inflammations may occur in another and much more evident manner, by means of a blood-clot in the veins, as we shall show in the section on thrombosis, embolism, and phlebitis. Non-traumatic inflammations may terminate in resolution, in firm organization of the inflammatory product, in suppuration, or in mortification. But we will now cease treating this subject in general terms, and pass to the inflammations of the different tissues.

## 1. ACUTE INFLAMMATION OF THE CUTIS.

The simple forms of acute inflammation of the skin (spots, wheals papules, vesicles, pustules), which are grouped under the common name of "acute exanthemata," belong to internal medicine. Only erysipelatous inflammation, furuncle, and carbuncle, are generally spoken of as true primary inflammations of the cutis. I will here remind you that very frequently the skin is secondarily affected, from inflammation of the subcutaneous cellular tissue and muscles, or even of the periosteum or bones.

(*a*.) Erysipelatous inflammation is located chiefly in the papillary layer and in the rete Malpighii. The local symptoms are great redness and œdematous swelling of the skin, pain on being touched, and subsequent detachment of epidermis; these are occasionally accompanied by very high fever, out of proportion to the extent of the local affection. The disease lasts from one day to three or four weeks. Any part of the skin or mucous membranes may be attacked, but idiopathic erysipelas is particularly frequent in the head and face Like the acute exanthemata of the skin, according to the views of many pathologists, erysipelas of the head and face should also be regarded as a symptomatic cutaneous inflammation; that is, that the local affection was only one symptom of an acute general disease. In that case, surgery would have as little to do with erysipelas as with scarlatina, measles, etc.; but, as it occurs especially in wounded persons, and particularly often around wounds, we must study it more attentively. I consider *erysipelas traumaticum* not as a symptomatic inflammation of the skin, but as a capillary lymphangitis of the skin, which is always due to infection. We shall treat of this disease more closely among the accidental traumatic diseases, and content ourselves here with having called attention to its relationship.

(*b*.) The furuncle or phlegmon is a peculiar form of inflammation of the skin, usually of typical course. Some of you may know it from personal observation. First, a nodule as large as a pea or bean forms in the skin; it is red and rather sensitive. Soon a small white point forms at its apex, the swelling spreads around this centre, and usually attains about the size of a dollar; sometimes the furuncle remains quite small, about the size of a cherry; the larger it is, the more painful it becomes, and it may render irritable persons quite feverish. If we let it run its own course, toward the fifth day the central, white point, becomes loosened in the shape of a plug, and pus mixed with blood and detached shreds of tissue is evacuated; three or four days later suppuration ceases, the swelling and redness gradually disappear, and finally only a punctate, scarcely-visible cicatrix remains.

We rarely have the opportunity of anatomically examining such furuncles in their first stage, as they are not a fatal disease; but, from what we see of the development and from incision, the death of a small portion of skin (perhaps of a cutaneous gland) seems to be the starting-point and centre of an inflammation, during which the blood finally stagnates in the dilated capillaries; by infiltration with plastic matter, the tissue of the cutis partly turns to pus, partly becomes gangrenous. The peculiarity in all this is, that such a point of inflammation should, as a general rule, show no tendency to spread, but should throughout remain circumscribed, and terminate with the detachment of the little plug above mentioned.

There is no doubt that in many cases the cause of single furuncles is purely local. Some parts where the secretion of the cutaneous glands is particularly strong, as the perinæum, axilla, etc., are especially predisposed to furuncles; they are also particularly common in persons who have very large sebaceous glands and so-called pimples, maggots, or comedones. But there are also undoubtedly constitutional conditions, diseases of the blood, which dispose to the formation of numerous furuncles on various parts of the body. This morbid diathesis is called *furunculosis;* should it continue long, it may prove very exhausting; the patients grow thin, and are greatly pulled down by pain and sleepless nights; children and weakly old persons may die of the disease. It is very popular to refer furuncles to full-bloodedness and fatness; it is believed that fatty food predisposes to them. In my country (Pomerania) they say that persons who suffer much from pustules and furuncles have "bad blood." I should very much doubt the truth of the supposition that fatty food especially disposes to furuncles. You will often find that miserable, atrophic children, and emaciated, sickly people, are frequently attacked by furuncle, and, although the lack of care of the skin has something to do with this. it is not the sole cause. On the other hand, it is also true that we nourished butchers are often attacked by furuncles; but this may be otherwise explained, for not unfrequently it may be found that in them the furuncles are due to poisoning by some animal matter; we should at least always examine for this cause. But I think it is going too far to assume that every furuncle is caused by infection, and is always to be regarded as one symptom of a general suppurative diathesis—of a pyæmia.

The treatment of individual furuncles is very simple. Attempts have been made to cut short the process, and prevent suppuration, by early applications of ice. But this rarely succeeds, and is a very tiresome treatment, which is not often popular with the patient. I prefer hastening suppuration by warm, moist compresses, and, if the furuncle does not spread too much, to quietly await the detachment of the central plug, then to squeeze out the furuncle, and do nothing more. If the furuncle be very large and painful, we may make one incision, or two crossing each other, through the tumor; then the natural course of the process is favored by the escape of blood, and the more rapid suppuration.

General furunculosis is a difficult disease to treat successfully, especially if we know little of its cause. Usually we give quinine, mineral acids, and iron, internally. Besides these, warm baths continued perseveringly are to be recommended. A perfectly-regulated diet, especially nutritious meats with good wine, is also advisable. The individual furuncles are to be treated as above advised.

(*c.*) *Carbuncle and carbunculous inflammation* (*anthrax*) anatomically resembles a group of several furuncles lying close together. The whole process is more extensive and intense, more inclined to progress, so that other parts may be affected by the extension of the inflammation. Many carbuncles, like most boils, are originally a purely local disease. Their chief seat is the hard skin of the back, especially in elderly persons. Their origin and first stage are the same as in furuncle. But soon a number of white points form near each other, and the swelling, redness, and pain, in the periphery, increase in some cases so much that the carbuncle may attain the size of a soup-dish; and, while the detachment of the white plugs of skin goes on in the centre, the process not unfrequently extends in the periphery. The detachment of gangrenous shreds is much greater in carbuncle than in furuncle. After the loss of the plugs of cutis, the skin appears perforated like a sieve, but subsequently not unfrequently suppurates, so that after a carbuncle a large cicatrix is always left. But, even when most intense, the process is almost always limited to the skin and subcutaneous cellular tissue; it is most rare for f
muscles to be destroyed, so that, when a large carbuncle

vicinity of an artery, the danger of destruction of the arterial walls is more apparent than real, as is shown by experience. After the extensive throwing off of the cellular tissue, and the final arrest of the process in the periphery, healthy and usually very luxuriant granulations develop; healing goes on in the usual manner, and is accomplished in a time corresponding to the size of the granulating surface.

You will have already noticed that the process of formation of furuncles and carbuncles differs from the inflammations with which you are already acquainted, by the constant and peculiar death of portions of skin; and I have mentioned that this gangrene of the skin, at first very small, is the primary and local cause of furuncles and carbuncles. Of course, this must be induced by an early, perhaps primary, occlusion of small arteries, possibly of the vascular net-work around the sebaceous glands, without our knowing on what final cause this latter depends.

The course of the ordinary carbuncle on the back is tedious and painful, although it rarely causes death. But there are cases, especially when the carbuncle or a diffuse carbunculous inflammation occurs in the face or head, which are accompanied by high fever and septic or, as was formerly said, "typhous" symptoms, and which prove dangerous and are even generally fatal (carbunculus maligna, pustula maligna). All carbuncles of the face are not of this malignant character; some run the usual course, and only leave a disfiguring cicatrix; but, as it is difficult and often impossible to tell how the case will turn out, I would advise you always to be very careful about the prognosis. Unfortunately, I have had such sad experience in these carbuncles of the face, that in any affection of the kind I am very solicitous about the life of the patient. Let me briefly narrate a case or two. In a young, strong, healthy man, on a journey to Berlin, from some unknown cause a painful swelling began in the lower lip; it increased rapidly, and soon spread to the whole lip, while the patient became very feverish. The surgeon who was called applied cataplasms, and apparently undervalued the condition of the patient, as he did not see him for two days. The third day the face was greatly swollen and the patient had a severe chill, and was quite delirious wh[illegible] to the clinic. I found the lip dark bluish-red with numer[illegible] grenous patches in the skin. Several incisions were made [illegible] wounds were dressed with chlorine-water, [illegible] bladder of ice placed on the head, as meningiti[illegible] soon as I saw the patient, I declared his condi[illegible] fell into a deep stupor, and died twenty-four [illegible] after the commencement of the carbuncle on [illegible] tunately, an autopsy was refused. I will mentio[illegible] student in Zürich received a sword-cut on the left side of the head. The wound healed without any remarkable symptoms; but it was a long while before it closed entirely. For some time there was a small, open wound, which was so slight that the patient paid no attention to it. Violent straining while fencing, and perhaps subsequently catching cold, may have been the causes of the following catastrophe. One morning the young man awakened with considerable pain in the cicatrix, and a general feeling of illness; a rosy redness and moderate swelling of the scalp rendered an attack of simple erysipelas capitis probable. But the fever increased in an unusual manner, without the redness spreading over the head. The patient had a chill, and became delirious. When on the third day he was brought to the hospital, in the vicinity of the cicatrix I found a number of small white spots, which showed me at once that there was carbunculous inflammation; as the patient was entirely unconscious, and for several reasons there was probably inflammation of the meninges of the brain, I had little hope of a cure; I gave the necessary directions, but the next day the patient was dead. The autopsy showed various white gangrenous points in the inflamed scalp cicatrix; on seeking further, the neighboring veins were found plugged with clots, and along them the cellular tissue was swollen and contained points of pus. Anteriorly I could follow this condition of the veins as far as the orbit, but did not try to follow it farther, not wishing to injure the eye. After opening the skull, as soon as the brain was removed, we found in the left anterior cranial fossa a moderately inflamed spot about as large as a dollar; the disease affected both the dura and pia mater, and even entered the brain-substance. There was no doubt that the inflammation starting from the cicatrix on the head had travelled along a vein into the cellular tissue of the orbit, and thence through the optic foramen and superior orbital fissure into the skull.

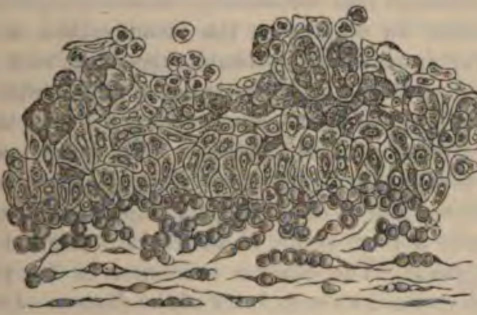
In many cases of malignant carbuncle of the face, on careful examination we shall find such an extension of the inflammation to the cranial cavity, and consequent disease of the brain. But I must remind you that the extent of this inflammation as found in the cadaver is not at all in proportion to the severity of the constitutional symptoms, so that the latter are by no means fully explained by the *post-mortem* appearances. Indeed, there are cases, and just the most quickly fatal ones, where death occurs without our being able to find any disease in the brain. Here there is full room for hypothesis; in the rapid, violent course and the quick change of carbunculous inflammation to gangrene we suspect a rapidly-occurring decomposition of the blood, of which the carbuncle itself may be either the cause or result. But, as the decomposition of the blood must have *its* cause, it has been supposed that an insect which has alighted on some carrion, or on the nose of a horse with glanders, or a cow with carbuncle, etc., lights soon after on a man and infects him; you will hereafter learn that malignant carbuncles result particularly from carbunculous cattle. I know of no cases where this has been actually observed, but I do not consider it impossible in certain cases; this supposition is supported by the fact that these carbuncles are most frequent on parts of the body which are usually exposed. At all events, the high fever and fatal blood-infection are mostly results of the local disease; hence, we must suppose that in these carbuncles, under circumstances which we do not exactly understand, peculiarly intense poisons are formed, whose reabsorption into the blood causes death. But the causes of malignant carbuncle are in most cases entirely obscure. In diabetes mellitus and uræmia carbuncle occurs, just as sugar is observed in the urine of persons otherwise healthy, who have furuncles and carbuncles; these are enigmatical facts. Fortunately, carbuncles are not frequent; even simple benignant carbuncles are so rare that in the extensive surgical policlinic of Berlin, where every year five or six thousand patients presented themselves, I only saw a carbuncle once in two years or so. In Zürich also they were rare. The *diagnosis* of ordinary carbuncle is not difficult, especially after you have seen one; diffuse carbunculous inflammation can only be recognized after a period of observation; at first it resembles erysipelas.

The treatment of carbuncle must be very energetic, if we would prevent the advance of the disease. As in all inflammations disposed to gangrene, numerous incisions should be made early, to permit the escape of the decomposed, putrid tissues and fluids. Hence in every carbuncle you make large crucial incisions, dividing the whole thickness of the cutis, and long enough to divide the infected skin clear through to the healthy. If this does not suffice, you add a few other incisions, especially where from the white points you recognize gangrene of the skin. The bleeding from these incisions is proportionately slight, as the blood is coagulated in most of the vessels of the carbuncle. In the incisions you place charpie wet with chlorine-water, and renew it every two or three hours; over this warm cataplasms may be regularly applied to hasten suppuration by the moist warmth. If the continued warmth be not well borne, as in carbuncle of the neck, where it may induce cerebral congestion, the cataplasms may be omitted and the antiseptic dressings continued alone, or even cold may be resorted to. If the tissue begins to detach, you daily pick off the half-loose tags with the forceps, and so try to keep the wound as clean as possible. Strong granulations will soon appear here and there; finally, the last shreds are detached and a honeycombed granulating surface is left; this soon smooths off, and subsequently cicatrizes in the usual manner, so that it only requires a little occasional stimulation from nitrate of silver, like other granulating surface. In malignant carbuncle the local treatment is the same that we have just described. For the rapidly-occurring cerebral disease the only thing we can do is to apply ice to the head. Internally we usually give quinine, acids, and other antiseptic remedies. But I must acknowledge that the results of this treatment are very slight, for in my own experience I do not know a case where it has succeeded in averting death when septicæmia was at all developed; this is the more depressing, because these malignant carbuncles generally attack young, strong individuals. Even if the course be favorable as regards life, there will be considerable loss of skin and great disfigurement, especially in carbunculous inflammation of the eyelids or lips, as they are mostly destroyed by gangrene. Early incision, excision, and burning out of the carbuncle, also have little effect on the further course of the disease, as I have proved to myself in a few malignant cases. But do not be deterred, by these hopeless views of treatment, from making early incisions, for cases occur where carbuncles on the face run the usual course after commencing with high fever. French surgeons have attained some good results by early burning out the malignant pustule.

## 2. ACUTE INFLAMMATIONS OF THE MUCOUS MEMBRANES.

While traumatic inflammation of the mucous membranes presents nothing peculiar, "acute catarrh" or "acute catarrhal inflammation" of these membranes is a peculiar form of disease which is anatomically characterized by great hyperæmia, œdematous swelling and free secretion of a fluid at first serous and subsequently muco-purulent, and is most frequently caused by catching cold or by infection. "Blennorrhœa" is an increase of catarrh to such a degree that quantities of pure pus are secreted. Catarrh and blennorrhœa may become chronic. Simple observation of exposed mucous membranes affected with catarrh shows that it may be very severe and long continued, without the substance of the membrane suffering much; the surface of the membrane is hyperæmic and swollen, somewhat thick and puffy; in rare cases there are superficial loss of epithelium and small defects of substance (catarrhal ulcers), but it is only in very rare cases that these cause more extensive destruction. This observation is supported by *post-mortem* examination and histological investigation. The opinion now is, that there is only a rapid throwing off of the epithelial cells which approach the surface as pus-cells, and that the connective-tissue layer of the mucous membrane takes no part in the process. Although many attempts have been made to find segregation of the cells in the deeper epithelial layers of mucous membranes affected with catarrh, they were unsuccessful till *Remak*, *Buhl*, and *Rindfleisch*, discovered large mother-cells in the epithelial layers of such membranes.

FIG. 62.



Epithelial layer of a conjunctiva affected with catarrh (after *Rindfleisch*). Magnified 400 diameters.

It was most natural to explain this observation by assuming that the mother-cells were formed by endogenous segregation of the protoplasm, and subsequently turned out their broods (as pus-cells). Since, in opposition to this view, it was repeatedly shown that, if this were the case, the mother-cells should always be found on catarrhal mucous membranes, while they were found only at first and then in small numbers, of late, they have been explained quite differently. *Steudener* and *Volkmann* first advanced the idea that the young cells do not form in the older ones, but that, under certain mechanically favorable influences, the latter may enter from without, but have nothing to do with the origin of the catarrh. Although this view is very difficult to prove, after much consideration and weighing of known facts, I consider it as very probable. This is not the place to go into details on the matter, but, since it has been proved by the cinnabar method that the white blood-cells escape from the vessels of the inflamed mucous membrane, and not only wander between the epithelium, but are also found as pus-cells in the catarrhal secretion I should think catarrhal pus had the same origin as other pus, viz. that it came directly from the blood. Besides catarrhal inflammation, mucous membranes are also subject to *croupous* and *diphtheritic* inflammations. When, in inflammation of a mucous membrane, the products of inflammation (cells and transudation) appearing on the surface form fibrine, and thus become a membrane clinging to the surface, which after a time dissolves into mucus and pus, or is lifted up by pus which is produced behind it from the mucous membrane, we call it a "croupous inflammation;" the mucous membrane and its epithelium meantime remain intact, the parts are perfectly restored. *Diphtheria* is exactly similar to the above process, but the fibrinous layer is not only attached more firmly to the tissue, but the serum permeating the substance of the membrane coagulates; the circulation is thus impaired so much that occasionally the affected part becomes entirely gangrenous. In diphtheria, the disintegration and gangrene are prominent symptoms; they probably depend on very rapid development of germs of fungi and infusoria in the diphtheritic membrane. Whether these fungous germs are, as many suppose, the cause of diphtheria, at present remains doubtful. The general affection, the fever, may be very severe in extensive croupous inflammation (as in the fine bronchi and alveoli of the lungs, croupous pneumonia), but in diphtheria it is of a more septic character; the latter disease is far the most malignant. The mucous membrane of the pharynx and trachea is often exposed to both forms of the disease. Catarrhal conjunctivitis, which is so very common, may become diphtheritic, but rarely becomes croupous. The mucous membrane of the intestinal canal is seldom the seat of these diseases, the same is true of the mucous membrane of the genitals, which are so often affected with contagious blennorrhœa (clap, gonorrhœa).

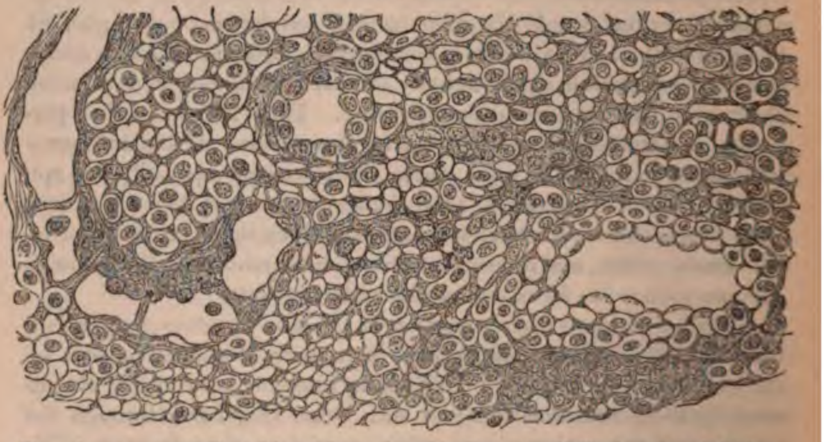
### 3. ACUTE INFLAMMATION OF THE CELLULAR TISSUE. PHLEGMONOUS INFLAMMATION.

This term is pleonastic, for ἡ φλεγμόνη means inflammation, but practically it is so exclusively applied to inflammation of the cellular tissue tending to suppuration, that every surgeon knows what it means; another name for the same disease is *pseudo-erysipelas;* it is just as much used, but seems to me less distinctive. The causes of this inflammation are in many cases very obscure; a severe cold can rarely be proved to be the cause; frequently these inflammations might result from infection, even if the cutis be uninjured, but this is only hypothesis; we have already seen these progressive acute inflammations as a complication in injuries, especially as a result of local infection from mortifying shreds of tissue in contusions and contused wounds. Spontaneous inflammation of the cellular tissue is most frequent in the extremities, more frequent above than below the fasciæ, especially prone to affect the fingers and hand; here it is called panaritium (corrupted from paronychia, inflammation around the nail, from ὄνυξ nail), and to distinguish it from deeper inflammations also occurring in the fingers and hand, panaritium subcutaneum. If the inflammation affect the vicinity of the nail, or the nail-bed itself, it is termed panaritium sub ungue. Let us first consider the symptoms of phlegmon of the forearm: it begins with pain, swelling, and redness of the skin, and usually with high fever; the skin of the arm is somewhat œdematous and very tense. With this commencement, which always announces an acute inflammation of the arm, its seat may vary greatly, and in the first day or two you may be unable to decide whether it is a case of inflammation of the subcutaneous cellular tissue, of perimuscular inflammation below the fascia, or even of periostitis or ostitis. The greater the œdema, the more considerable the pain, the less the redness of skin, and the less intense the fever, the more probably you have to anticipate a deep-seated inflammation which will terminate in suppuration. If the inflammation attacks only the subcutaneous cellular tissue, and goes on to suppuration, as it does in most cases (though resolution is seen), this evinces itself in a few days by the skin becoming red at some point, and distinct fluctuation occurring. Then the pus either perforates spontaneously, or is let out by an incision. If the inflammation affect parts of the body where the skin, and especially the epidermis, is particularly thick, as in the hands and feet, there is at first little perceptible redness, as it would be hidden by the thick layer of epidermis. Pain, and a peculiar tension and throbbing in the inflamed part, announce the formation of pus under the skin.

In some of these cases a portion of the skin becomes gangrenous, the circulation being disturbed by the tension of the tissue, part of the skin loses its vitality. The fasciæ also are occasionally threatened by these inflammations; in such cases they come through the openings of the cutis as large, white, consistent, thready tags. This is particularly the case in inflammations of the scalp, which not un frequently extend over the entire skull; the whole galea aponeurotica may thus be lost.

Let us now pass to the more minute anatomical changes that take place in acute inflammation of the cellular tissue; we shall not here return to the dispute as to whether vessels, tissues, or nerves, are first affected, but shall only speak of what we can find on direct anatomical examination. A series of observations on the cadaver, where in various cases we see inflammation in different stages, gives us sufficient information on this subject. The first things we find are distention of the capillaries and swelling of the tissue by serous exudation from the vessels, and a rich, plastic infiltration, varying with the stage, i. e., the connective tissue is filled with quantities of young, round cells. This, then, is the anatomical condition of the cellular tissue under the œdematous, reddened, painful skin; subsequently the collection of cells in the inflamed connective tissue and fat becomes more prominent. These tissues become tense, and there is stagnation of blood in the vessels at various points, especially in the capillaries and veins; at some places the circulation ceases entirely. This stagnation of the blood, which at first causes a dark-blue color, and then whiteness from the rapid discoloration of the red blood-cells, may extend so far as to cause extensive gangrene of the tissue, a result which we have already mentioned. But in most cases this does not occur, but while the cells increase, the fibrillar intercellular substance disappears, partly by the death of small tags and particles, partly by gradually becoming gelatinous, and finally changing to fluid pus.

FIG. 63.



Tissue from a prepuce infiltrated from inflammation. The filamentary fibrillar formation of the tissue has entirely disappeared, from the softening influence of the cellular inflammation. The walls of the vessels are relaxed and perforated. Magnified about 500.

As the inflammation progresses the entire inflamed part is finally changed to pus, that is, to fluid tissue, consisting of cells with some serous intercellular fluid which is mixed with shreds of dead tissue. If the process goes on in the subcutaneous cellular tissue, extending in all directions (most rapidly where the tissue is most vascular and richest in cells), the purulent destruction of tissue or suppuration will extend to the cutis from within, perforate it at some point, and through this perforation the pus will escape outwardly; when this occurs, the process often ceases to extend. The tissue surrounding the purulent collection is filled with cells and very vascular; anatomically it closely resembles a granulating surface (without any distinct granulations) lining the whole cavity. When the pus is all evacuated the walls come together and usually unite quickly. The plastic infiltration continues for a time, causing the skin to remain firmer and more rigid than usual. But, by disintegration and reabsorption of the infiltrating cells, and transformation of the connective-tissue substance, this state also returns to the normal.

You will readily perceive that, anatomically, the process is much the same whether it be diffuse or circumscribed; the finer changes of tissue in the two are just the same. But in practice we distinguish between *purulent infiltration* and *abscess*. The first expression explains itself: by an abscess we usually understand a circumscribed collection of pus, excluding further progress of the inflammation; those forming rapidly, from acute inflammation, are called acute or *hot abscesses*, in contradistinction to *cold abscesses*, or those due to chronic inflammation. The following figure (Fig. 64) may render the formation of abscess more clear to you.

You here see how the young cells gradually collect at the points where the connective-tissue corpuscles lay, while intermediate substance constantly decreases, and how in the middle of the drawing, in the centre of the inflamed spot, the groups of cells unite and form a collection of pus; every abscess at first consists of such separate collections of pus; it grows by peripheral extension of the suppuration. Formerly, it was not doubted that, wherever pus-cells thus appeared in groups, they were to be regarded as a production of connective-tissue cells; according to our present views, there is no doubt that these young cells are escaped white blood-cells, and are simply grouped

FIG. 64.

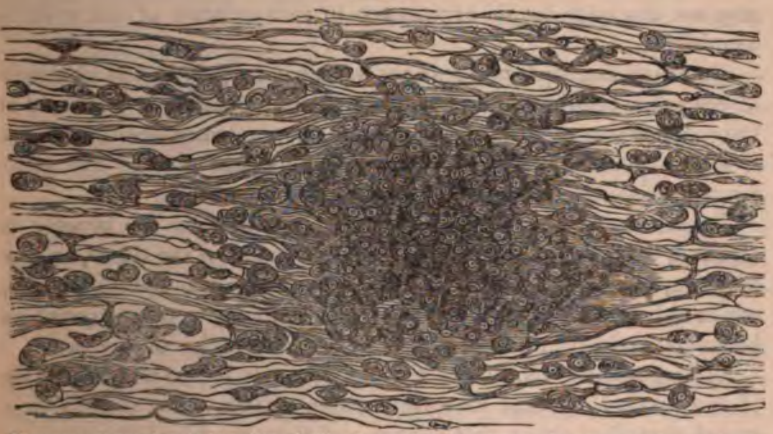
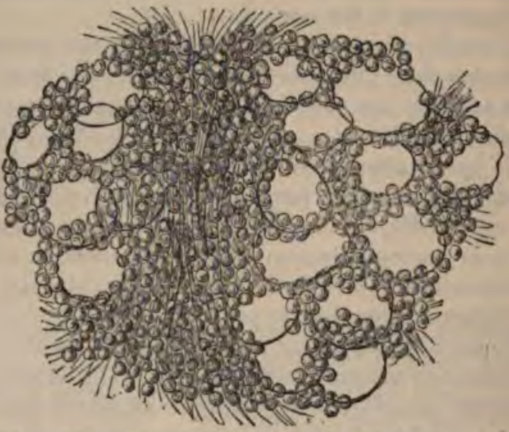


Diagram of purulent infiltration of the cutis connective tissue, forming an abscess in the middle. Magnified 350 diameters.

together from mechanical causes. The fat, which is usually plentiful in the subcutaneous cellular tissue, is generally destroyed in acute inflammation, the fat-cells being compressed by the new cell-masses, and the fat becoming fluid; subsequently, it is occasionally found in the shape of oil-drops mixed with the pus. In this preparation you may see the microscopic appearance in inflammation of the cellular membrane.

FIG. 65.



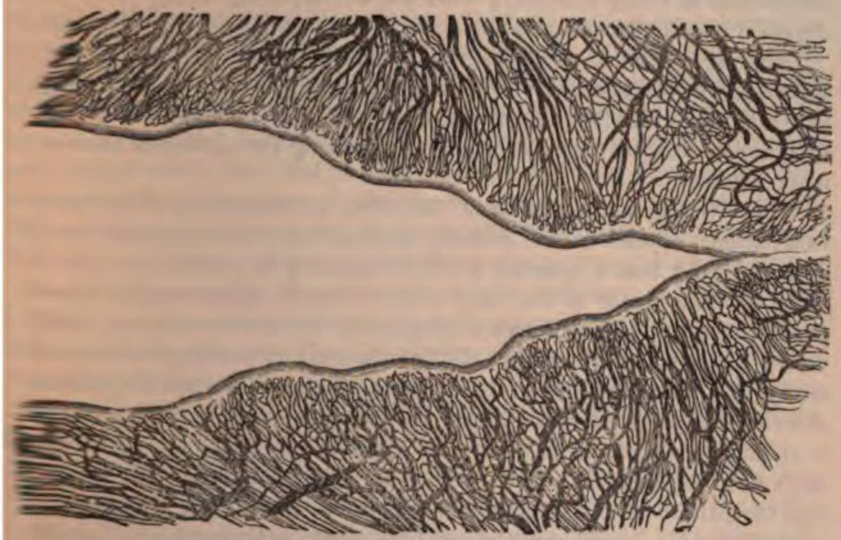
Purulent infiltration of the cellular membrane. Magnified 350 diameters; from a preparation hardened in alcohol.

In examining such preparations we not unfrequently find filaments of coagulated fibrine infiltrated in the tissue; possibly it is formed at the commencement of the inflammation, as previously described; but it is also possible that these filaments appertain only to the fully-formed pus—possibly they are produced by the alcohol.

I must call your attention to the fact that, until the process is arrested, we always have a progressive softening of the tissue, or suppuration, in which it differs from a developed granulating surface, which only forms pus on its surface. All suppurative parenchymatous inflammations have a destructive or deleterious action on the tissue.

As regards the relation of the blood-vessels to the new formation of the young tissue and its speedy disintegration, it has already been stated that they are at first dilated, and then the blood stagnates in them; if the circulation be entirely arrested in certain portions of tissue, in which case the coagulation in the veins occasionally extends a considerable distance, the walls of the vessels and the clot suppurate, or fall into shreds, as far as the border where the circulation

FIG. 66.



Vessels (artificially injected) of the walls of an abscess that had been induced in the tongue of a dog. Magnified 25 diameters.

begins again. As we have already seen when studying the detachment of necrosed shreds of tissue, vascular loops must form on this border of the living tissue; that is, the whole inner surface of an abscess, in the arrangements of its vessels, is analogous to a granulating surface folded up sac-like.

In regard to the lymphatic vessels, we may conclude from analogy that here, as in the vicinity of wounds, they are closed by the inflammatory neoplasia; special investigations on this subject would be very desirable. So soon and so long as an abscess is surrounded by a vigorous layer of tissue infiltrated with plastic matter, for reasons already mentioned there will be no reabsorption of purulent or putrid substances from the cavity of the abscess. I can give you practical evidence of this, if in the clinic you will smell pus from an abscess near the rectum or in the mouth; this pus has an exceedingly penetrating, putrid odor, still is not reabsorbed by the walls of the veins, or is so to only a very slight extent; symptoms of general sepsis very rarely occur. But at the commencement of inflammation, and later, when it is accompanied by *rapid* destruction of tissue, as well as in some progressive inflammations around contused wounds, and in phlegmonous inflammation of the cellular tissue, etc., if the lymphatic vessels are not yet stopped by cell-formation, organized inflammatory new formation does not occur, or comes on late as the gangrenous destruction is being bounded; then the decomposing tissue enters the open lymphatics and acts as a ferment in the blood, causing fever.

Although inflammation of the cellular tissue (cellulitis) may occur at any part of the body, it is most frequent in the hand, forearm, knee, foot, and leg. It is often accompanied, and, when extending, preceded, by lymphangitis, of which we shall speak among the accidental traumatic diseases.

The intensity and duration of the fever, accompanying these inflammations, depend on the quantity and quality of the material reabsorbed. At first a quantity of these matters is thrown into the blood at once, hence at the onset there is usually high fever, sometimes chill; as the inflammation progresses, the fever continues; it ceases when further absorption of the inflammatory product is arrested by the above changes of tissue, when the process stops and the abscess is formed. The quality of the inflammatory material formed in cellular inflammation certainly varies greatly; for instance, in some cases deep in the neck in old people there is such intense poisoning that the patients die without other symptoms. It is here the same as in carbuncle—some cases cause little fever, others produce fatal septic fever. If a phlegmon be due to a dangerous poison, such as that of glanders, we do not wonder at the fatal termination; but for the spontaneous cases it often seems very strange why some should be so very severe, while most of them are relatively mild.

The prognosis of phlegmonous inflammations varies immensely with the location, extent, and cause. While the disease, occurring as a metastasis in a general phlogistic or suppurative diathesis, or in glanders, gives little hopes of cure, while deeply-seated abscesses in the walls of the abdomen or in the pelvis are very slow in their course and may prove dangerous from the locality, or, by destruction of fasciæ, tendons, and skin, may impair the functions, most cases of phlegmon on the fingers, hand, forearm, etc., are only moderate diseases of short duration, although very painful. The sooner suppuration occurs and the more circumscribed the inflammation, the better the prognosis

As regards the *treatment*, at the commencement of the disease its aim is to arrest the development of the disease if possible, that is, to attain the earliest possible reabsorption of the serous and plastic infiltration. For this purpose there are various remedies: first, the external use of mercury; the inflamed part may be smeared with mercurial ointment, the patient placed in bed, and the inflamed extremity enveloped in warm, moist cloths or large cataplasms. Ice also may be employed at first, if the whole inflamed part can be covered with several bladders of ice. Compression by adhesive plaster and bandages is also a very effective remedy for aiding absorption, but it is little used in these inflammations, partly because of the pain it causes in such cases, partly because the remedy is not free from danger, as gangrene may be easily induced by a little too much pressure. If the process be not moderated soon after the employment of the above remedies, but all the symptoms increase, we must give up the hope of resolution, and resort to remedies to hasten the suppuration which we cannot avert; the chief of these is the application of moist warmth, especially in the shape of cataplasms. Then, as soon as fluctuation is detected at any point, we do not usually leave the perforation to Nature, but divide the skin to give vent to the matter; if the suppuration extends under the skin, we make several openings, at least I prefer this to one very large incision, from the elbow to the hand for instance, because in the latter the skin gapes widely, and takes a long time to heal. If the pus escapes naturally from the openings, great cleanliness is the only thing necessary; this is greatly assisted by local warm baths.

While it is a very simple thing to open subcutaneous abscesses, "oncotomy" of deep abscesses requires great attention to the anatomy of the locality: for instance, the diagnosis may be very difficult in suppurations deep in the neck, in the pelvis, in the abdominal wall, etc., and can only be certainly made after a long period of observation; still, partly for the relief of the patient, partly to avoid a spontaneous opening into the abdomen, perhaps it may be desirable to evacuate the pus early. In such cases we must not plunge a bistoury boldly in, but dissect up layer after layer, till we reach the fluctuating covering of the abscess; then introduce a probe carefully, and dilate the opening by extending the blades of forceps introduced into it, so as to avoid hæmorrhage from the deeper parts. Occasionally decomposition of the pus in an abscess causes so much gas as to give rise to a tympanitic percussion-sound; after being opened, these putrid abscesses should be syringed out and dressed with chlorine-water.

### 4. ACUTE INFLAMMATION OF THE MUSCLES.

Idiopathic acute inflammation of muscular substance is relatively rare. It occurs in the muscles of the tongue, in the psoas, pectoral, and gluteal muscles, and in those of the thigh and calf of the leg; the usual termination is in abscess, although resolution has been observed. Metastatic muscular abscesses are very frequent in glanders. Regarding the special histological conditions, the interstitial connective tissue of the muscles, the perimysium is here, as in traumatic myositis, the chief seat of the purulent infiltration; from the very acute disease, the nuclei of the muscular filaments are destroyed, with the contractile substance and the sarcolemma; only on the stumps of the muscular filaments in the capsule of the abscess do we find the muscular nuclei (muscular corpuscles) in groups and adherent to the cicatrix; in such cases, according to *O. Weber*, there is a considerable new formation of young muscle-cells. The symptoms of an abscess in the muscle are the same as those of any deep abscess; their periods of development and perforation vary with their size and extent. In many cases there is contraction of the muscles in whose substance the abscess develops, as in psoitis. I shall not discuss whether this is the physiological result of the inflammatory irritation, or whether it is half voluntary, and made instinctively by the patient, but am rather inclined to the latter view, for in small and not very painful abscesses and in traumatic inflammations of the muscles, there is usually no contraction, but this occurs only in large abscesses, which are compressed by strong fasciæ. Abscesses in muscles should be opened as soon as fluctuation is felt, and the diagnosis certain.

A very peculiar form of disease of the muscles, which, according to my view, should be classed among subcutaneous inflammations, has been recently discovered and described by *Zenker;* it occurs chiefly in typhoid fever, in the adductor muscles of the thigh; in it the contractile substance in the sarcolemma crumbles and is gradually absorbed, while new muscular filaments form to replace the old. Thus, in most cases, the parts are fully restored; in other cases permanent atrophy of the muscle remains. There is no accurate knowledge as to whether this disease may lead to suppuration, although abscesses of the abdominal muscles have been observed after typhus.

### 5. ACUTE INFLAMMATION OF THE SHEATHS OF TENDONS AND SUBCUTANEOUS MUCOUS BURSÆ (SEROUS MEMBRANES).

As is well known, the sheaths of tendons form shut sacs, which enclose some of the tendons of the hands and feet. They may become acutely inflamed from contusion, and in some few cases also spontaneously. Like all acutely-inflamed serous membranes, these sacs at first exude a quantity of fibrinous serum; recent fibrinous pseudo-membranes composed of wandering cells may again dissolve, but they may also induce temporary or permanent adhesions of the sheath to the tendon; lastly, there is not unfrequently suppuration of the membrane, and at this time the tendon may become necrosed. Pain on motion and slight swelling are the first signs of such inflammation; occasionally there is friction-sound, a grating in the sheath of the tendon, which may be perceived by the hand, or, still better, by the ear. This noise is due to the surfaces of the tendon and of its sheath having become rough from deposits of fibrine and rubbing against each other, when the tendons are moved; this form of subcutaneous inflammation is most common on the back of the hand, and almost always terminates in resolution. The very acute inflammations of the sheaths of the tendons, arising from unknown causes and going on to suppuration, are rare; they begin like an acute phlegmon; the subcutaneous cellular tissue quickly participates in the inflammation; the limb swells greatly, and the adjacent finger or wrist-joint may be drawn into the inflammation. Like the synovial membrane of the joints, that of the tendinous sheaths occasionally seems to furnish products that intensely affect the surrounding parts. If, under suitable treatment, the disease does not go on to suppuration, or, if this be only partial, resolution slowly occurs; the limb remains stiff a long while; the adhesions between the tendon and its sheath do not break down till after months of use. If there be extensive suppuration of the sheaths of the tendon (which, in the hand, has been termed "panaritium tendinosum"), the tendons usually become necrosed, and after a time may be drawn out of the abscess openings as white threads and shreds; the membrane then degenerates to spongy granulations. If the process be now arrested, one or more fingers will be stiff, and remain so for life. If the joints be also attacked in the fingers, there may be recovery with anchylosis; but, if the wrist or ankle-joint be affected, its existence will be greatly endangered. In acute suppurative inflammation of the tendinous sheaths, the *fever* is occasionally slight at first, but in severe cases the disease may begin with a chill. The further the inflammation and suppuration extend, the less the process tends to formation of an abscess, the more continued the fever becomes, and it assumes a distinctly remittent form; at the same time the patients are rapidly pulled down; in a few weeks the strongest men emaciate to skeletons. The prognosis is bad when the fever runs on with intermittent attacks and chills.

The *treatment* of subcutaneous, crepitating inflammations of the sheaths of the tendons consists in keeping the part quiet on a splint, and painting it with tincture of iodine; if this does not afford speedy relief, a blister may be applied; under this treatment I have always seen this form of inflammation disappear in a few days. If the symptoms are severe from the first, quiet of the part is the first requisite; this should be seconded by mercurial ointment and bladders of ice. This treatment should be persistently pursued; in these cases I decidedly prefer it to cataplasms and local warm baths, which are very common. If absceses form, incisions and plenty of counter-openings should be made; in these cases drainage-tubes are very useful, because the granulations projecting from the openings often obstruct the escape of the pus. If the suppuration will not stop, if the spongy swelling of the limb continues, if crepitation appears in the joint between the bones of the wrist (showing that the cartilaginous coverings have suppurated), and if the patient continues to sink, there is little hope of a termination in anchylosis of the hand, but the danger to life is so great that amputation of the forearm should be made; the patient may thus escape with his life, and will soon recover his strength.

Acute inflammations of the *subcutaneous mucous bursæ* are less dangerous; the bursa præpatellaris and anconea are most frequently affected either from injury or spontaneously; they are connected neither with the joint nor with the sheaths of the tendons; they become painful, fill with fibrinous serum, the skin reddens, and the cellular tissue in the vicinity participates in the inflammation; but suppuration rarely occurs if the patient is treated early. The remedies are mercurial ointment or tincture of iodine, keeping the limb quiet, and compressing the swollen bursa by applying wet bandages. Puncture is unnecessary, and may be injurious, from being followed by suppuration and a tedious suppurating fistula.

Limitation of carbuncle to the skin and subcutaneous cellular tissue is very characteristic of fibrinous (diphtheritic) inflammations; so that on this account, as well as from the hard infiltration and necrosis of the tissue once infiltrated, I do not hesitate to consider carbuncle as a diphtheritic inflammation of the skin. I have had no opportunity of examining to see if there are micrococci in the freshly-expressed juice of carbuncle; finding a few of them in the exposed shreds of necrosed tissue would prove nothing about the origin of the carbuncle. [In diphtheria of mucous membranes, as well as in that of wounds, the infiltrated tissue contains micrococci constantly multiplying. It was long disputed whether these were merely accidental accompaniments or the actual exciters of the disease. Now it seems proved that by cultivation in proper fluids they may be separated from the organic decomposing products clinging to them; that after inoculation in living tissue there is active increase; that the fungi enter the tissue, and by their mechanical and perhaps chemical irritation induce a peculiar inflammation, characterized by the coagulating transudation permeating the tissues. Chemical products of very poisonous character form, cling to the micrococci, and are carried along with them. The more rapidly these substances are absorbed, the larger the amount absorbed, the more intense the constitutional effect.] *Kochmann* thinks that carbuncle as well as furuncle originally develops from a sweat-gland, or from several adjacent glands. *J. Neumann* distinguishes between carbuncles from sweat-glands and from cellular tissue. I cannot say whether there is any justice in this distinction, as I too rarely see carbuncle in its first stages.

The inflammation described (p. 308) cannot be considered exactly as carbuncle; it is rather a carbunculous inflammation of the skin and subcutaneous tissue, which I should now prefer to call diphtheritic phlegmon; the accompanying erysipelatous redness also corresponds with diphtheritis.

The great difference of the constitutional symptoms in carbuncles agrees very well with the supposition that they are of diphtheritic nature, where it is characteristic for the local extent not to correspond to the general toxic symptoms. I do not know if paralyses ever occur after carbuncle, as they do after pharyngeal and laryngeal diphtheria.

You will often hear that early incisions in phlegmonous inflammation will prevent the skin from becoming gangrenous or suppurating. Unfortunately, I cannot confirm this. I have found it to depend more on the intensity of the inflammation than on the tension of the skin. Still, I consider early incisions proper in phlegmonous inflammation, as it seems that by carefully pressing the serum out of the inflamed tissue we may sometimes arrest the progress of the affection.

# CHAPTER XI.

## *ACUTE INFLAMMATIONS OF THE BONES, PERIOSTEUM, AND JOINTS.*

### LECTURE XXII.

Anatomy.—Acute Periostitis and Osteomyelitis of the Long Bones: Symptoms, Terminations in Resolution, Suppuration, Necrosis, Prognosis, Treatment.—Acute Ostitis in Spongy Bones.—Acute Inflammations of the Joints.—Hydrops Acutus; Symptoms, Treatment.—Acute Suppurative Inflammations of Joints: Symptoms, Course, Treatment, Anatomy.—Acute Articular Rheumatism.—Arthritis.—Metastatic Inflammations of Joints (Gonorrhœal, Pyemic, Puerperal).

THE periosteum and the bones are physiologically so intimately connected that disease of one generally affects the other; although, in spite of this, we are, for practical reasons, obliged to consider acute and chronic inflammation of the periosteum and of bone separately, still we shall often have to refer to their connection. I must here make a few preliminary anatomical remarks, as they are important for the comprehension of the following process: When speaking briefly of the periosteum, we usually mean, simply, the white, glistening, thin membrane, poor in vessels, which immediately surrounds the bone. I must here remark that this represents only a part of the periosteum that is pathologically of little relative importance. Upon this just described inner layer of the periosteum lies, at points where no tendons or ligaments are inserted, a layer of loose cellular tissue, which is also to be considered as periosteum, and in which principally lie the vessels that enter the bone. This outer layer of periosteum is the most frequent seat of primary inflammations, either acute or chronic; the loose cellular tissue of which this layer consists is very rich in cells and vessels, hence more inclined to inflammation than is the tendinous portion, poor in cells and vessels, which lies immediately on the bone. As to nutrient vessels, especially in the long bones, the epiphyses have their own supply, which, as long as the epiphyseal cartilages continue, do not communicate with the vessels of the diaphysis, which have their own nutrient arteries. This distribution of the vessels explains why diseases of the diaphyses in young persons rarely pass to the epiphyses and the reverse. Genetically the articular capsule is a continuation of the periosteum, and a certain connection is often observed between articular and periosteal diseases, the diseases of one readily passing to the other. In the course of the following observations we shall have occasion to recur to these anatomical conditions.

First, let us speak of *acute periostitis* and *osteomyelitis*, of which you have already heard something in the remarks on suppuration of bone in the chapter on open fractures (p. 211). This disease is not very frequent; it occurs chiefly in young persons, and in its typical forms almost exclusively in the long bones. The femur is most frequently attacked, next the tibia, more rarely the humerus and bones of the forearm. I have seen the disease occur primarily or secondarily in the vicinity of acutely-inflamed joints, after catching cold, and after severe concussions and contusions of the bones. It is possible that the extravasation into the medulla from crushing or contusion of a bone may be reabsorbed, without the occurrence of any symptom but a continued pain as the result of the injury; but such injuries may occasionally induce chronic affections of various sorts.

In many cases we cannot discover whether only the periosteum or the medulla of the bone is affected; the distinction is usually only rendered certain by the subsequent course and by the termination. The symptoms are as follows: The disease begins with high fever, not unfrequently with a chill; there is severe pain in the affected limb, which swells at first without redness. The severe pain prevents motion of the limb; every touch or the slightest jarring is very painful; the skin is tense, usually œdematous, and occasionally the distended subcutaneous veins show through, a sign that the flow of blood to the deeper parts is obstructed. The inflammation may affect the whole or only part of a bone. But these symptoms simply indicate the existence of an intense deeply-seated acute inflammation. But as idiopathic inflammation of the perimuscular and peritendinous cellular tissue is very unfrequent, and rarely begins with so much pain, we shall not err in most cases if, with the above symptoms, we diagnosticate acute periostitis, perhaps accompanied by osteomyelitis. If, while there are great pain and fever, or complete inability to move the limb on account of pain, swelling does not occur for several days, we may suspect that the primary seat of the inflammation is the medullary cavity of the bone, and that at first the periosteum participates but little. In this stage the diseased part is in about the following condition: The vessels of the medulla and periosteum are greatly dilated and distended with blood; perhaps there may be stasis of blood at different points. The medulla, instead of its usual bright-yellow color, is dark blue, and permeated with extravasations; the periosteum is greatly infiltrated, and on microscopical examination of it you find numbers of young cells, as you also do in the medulla; that is, there is plastic infiltration. In this stage, a complete return to the normal state is possible, and, if proper treatment is begun early, this is not so rare, particularly in the more subacute cases. The fever falls, the swelling decreases, and the pain ceases; a fortnight after the commencement of the disease the patient may be recovered. Even when the process is somewhat further advanced, it may stop; then a part of the new formation on the surface of the bone ossifies, and thus, for a time at least, there is thickening of the affected bone, which may again be absorbed in the course of months.

In most cases the course of periostitis is not so favorable, but the process goes on, and terminates in suppuration, the symptoms being as follows: The skin of the swollen, tense, and painful limb is at first reddish, then brownish red; the œdema extends further and further; the neighboring joints become painful, and swell; the fever remains at the same point; the chills are not infrequently repeated. The patient is much exhausted, as he eats little, and at night is kept awake by the pain. Toward the twelfth or fourteenth day of the disease, rarely earlier, but often later, we may clearly distinguish fluctuation, and may then greatly alleviate the sufferings of the patient by letting out the pus through one or more openings, if the skin over the abscess is sufficiently thinned; for the opening of deep, stiff-walled abscesses which do not collapse may prove dangerous from decomposition of blood and pus in the insufficiently-encapsulated abscess. The spontaneous perforation, especially the suppuration of the fascias, occasionally takes a good while, and, moreover, the openings thus formed are usually too small; they must subsequently be enlarged. If you introduce the finger through one of these artificial openings, you come directly on the bone, and in many cases find it denuded of periosteum. The extent to which this denudation occurs depends on the extent of the periostitis. It may extend the whole length of the diaphysis, and in these worst cases the symptoms are the most severe. Probably, however, only a half or a third of the periosteum is diseased, nor is the entire circumference of the bone necessarily affected, but perhaps only the anterior, lateral, or posterior portion is so. The periostitis is particularly apt to stop at the points of origin or insertion of strong muscles. In those cases of slight extent all the symptoms will be milder.

Even in this stage the disease may take one of two different directions: possibly, after the evacuation of the pus, the soft parts may quickly become adherent to the bone, as the walls of an acute abscess do to each other. I have seen this a few times in periostitis of the femur in children two or three years old. After the opening, a slight quantity of pus continued to discharge for only a short time. The openings soon closed entirely, the tumor receded, and perfect recovery took place. But, according to my experience, such a termination only occurs in small children. More frequently, as a result of the suppuration of the periosteum, the bone is mostly robbed of its nutrient vessels, and partly or wholly dies, leaving the condition termed necrosis, or gangrene of the bone. The extent of this necrosis will essentially depend on the extent of the periostitis. The partially or entirely destroyed diaphysis of the long bones must be detached as a foreign body, as we have seen to be the case in gangrene of the soft parts and traumatic necrosis. This requires a long time; hence the process of necrosis, the detachment of the portion of dead bone or sequestrum, and every thing connected with it, is always a chronic one. We shall have to speak of this hereafter. Before the inflammation passes into this chronic state, acute suppuration continues for a time after the first opening of the abscess. Various complications, even pyæmia, may occur. Whenever these patients are feverish, they are in danger.

We must again return to the medulla of the bone, which we left in the first stage of inflammation. Here, also, the inflammation may terminate in suppuration. If the osteomyelitis be diffuse or total, the whole medulla may suppurate. This suppuration may even assume a putrid character, and induce septicæmia. If there be extensive suppurative osteomyelitis, with suppurative periostitis, death of the diaphysis of the bone is certain. Should there be only partial suppuration of the medulla, or if there be none at all, the circulation of blood in the bone may be preserved and the bone remain viable. It may not infrequently occur that, under such circumstances, the bone will waver for a time between life and death, as the feeble circulation nourishes the bone very incompletely. Acute suppurative osteomyelitis, without participation of the periosteum, probably does not occur; it is not infrequently combined with *osteophlebitis*, which may end in putrefaction or suppuration of the thrombus, and is prone to induce metastatic abscesses. Another not infrequent, though not constant, accompaniment of osteomyelitis is suppuration of the epiphyseal cartilages in persons in whom they still exist, that is, till about the twenty-fourth year. The process is not difficult to explain. The suppuration may extend to the epiphyseal cartilage partly from the medulla of the bone, partly from the periosteum. If it suppurate, the continuity of the bone is destroyed, and at the seat of the epiphysis there is motion, as in fracture; dislocations may also be caused by contraction of the muscles. Usually there is only *one* such epiphyseal separation of the affected bone, above or below; in rare cases it is double. I have once seen this double separation of the epiphyses in the tibia; several times I have seen separation of the lower epiphysis of the femur, once of the upper end of this bone, once of the lower end of the humerus, twice of the upper end. In one case I saw epiphyseal softening, with luxation of the lower end of the femur, occur without suppuration. It has already been stated that inflammation of the neighboring joints are apt to accompany periostitis. These articular inflammations usually have a rather subacute course. The serous fluid collecting in the joint is usually reabsorbed as the acute disease of the bone subsides, but the joint often remains swollen, and not infrequently permanently stiff. Several times, also, I have seen acute periostitis and osteomyelitis of the femur succeed acute articular rheumatism of the knee. Lastly, we must also mention that this osteomyelitis may occur in several bones at once.

The diagnosis as to how far periosteum and bone are affected in the acute disease cannot be made with any certainty, but can only be decided by the extent of the consequent necrosis; and even this is no accurate measure, for the periostitis may end in suppuration, while the inflammation in the bone may end in resolution, or only cause some interstitial formation of bone. The process may start: 1. In the loose cellular-tissue layer of the periosteum; this suppurates. If the suppuration be limited to this layer, after opening the abscess we may pass the finger directly to the surface of the bone, which we find covered with the granulating tendinous part of the periosteum; if the latter layer also suppurates, as it not infrequently does, the bone lies exposed, and the suppuration may continue into it. Thus osteomyelitis accompanies periostitis. If it be denied that the loose cellular layer is periosteum, but is to be regarded as part of the intermuscular cellular tissue (which would not be natural, because the vessels escaping from the bone lie chiefly in this layer), then there is no such thing as acute periostitis; for the tendinous portion of the periosteum is as little liable to primary inflammation as the fascias and tendons. 2. The inflammation begins in the bone, and thence extends to the periosteum and cellular tissue; osteomyelitis is the primary, periostitis the secondary, disease. Then there is pus not only in the bone, but on its surface, close under the tendinous portion of the periosteum. This is elevated by the pus, as far as its elasticity permits; it is then perforated, and the pus escapes into the cellular tissue

Here it causes more suppuration, and thus the process advances to the surface. *Roser* asserts that in these cases fluid fat is pressed, by the strong arterial pressure, from the cavity of the bone through the Haversian canals of the cortical substance to the surface of the bone, so that we may diagnose osteomyelitis from pus mixed with fat-drops rising from under the periosteum. Moreover, in a few cases, *Roser* found a remarkable elongation of the bone, and a relaxation of the neighboring joints, after osteomyelitis. He refers this to too rapid growth of the articular ligaments and epiphyseal cartilages.

In the *prognosis* of acute periostitis and osteomyelitis we have to distinguish between the danger to the existence of the bone and to life. If the disease induces partial or total necrosis of the bone, the disease may be very protracted; it may last several months, or even years. Acute periostitis and osteomyelitis, especially in the femur, and still more when double, is always dangerous to life, because pyæmia is so apt to occur, and in children, because of the profuse suppuration, it is the more dangerous the longer the condition remains acute and the further it spreads.

In treating this disease we may accomplish more if we are called early; one of the most efficient remedies is painting the whole limb with strong tincture of iodine. This remedy should be continued till large vesicles form. Of course the patient is to be kept recumbent, which in most cases does not need to be urged, as the pain keeps him quiet. Since commencing this treatment I am so well satisfied with it, that I have almost given up the other antiphlogistics; cups, leeches, mercurial ointment, etc. When the vesicles formed by the iodine dry up, you apply more. Derivation to the intestinal canal by saline purgatives aids the treatment, as it does in all acute inflammations. Some surgeons greatly praise the local application of ice at the commencement of the disease. Should suppuration nevertheless occur, and distinct fluctuation be felt at the thinnest part of the skin, we may make several openings in such a way that the pus shall escape without being pressed out; then the swelling usually subsides quickly; it is most favorable when the fever ceases early and the disease becomes chronic. If the fever continues, the suppuration remains profuse, the pains do not cease. We may try to relieve this condition by continued applications of bladders of ice, with which we also try to alleviate any inflammations of the joint that may occur. I have also derived great advantage from the application of a fenestrated plaster-splint, which should be supported with hoops on account of the large openings that must be made in it; in cases where there is detachment of the epiphysis, it is absolutely necessary that the limb should be fixed, if only to render the daily dressing less painful

Many surgeons do not follow this treatment, which is backed by a series of favorable cases. Some recommend making large, deep incisions down to the bone at the very start, or at least as soon as suppuration begins. Such extensive wounds are bad in feverish patients; I am satisfied that, under these circumstances, this heroic treatment renders the condition worse, it increases the predisposition to pyæmia. The idea that in acute osteomyelitis exarticulation should be made at once, as otherwise pyæmia is unavoidable, seems to me even more erroneous. This belief is certainly untrue, and under such circumstances amputation is not indicated, first, because at the onset the diagnosis of osteomyelitis is not absolutely certain, as the case might possibly be one of simple acute periostitis; secondly, because the prognosis in exarticulation of large limbs, if done for acute disease of the bone, is always very doubtful. In acute periostitis and osteomyelitis, of the tibia for instance, I should only amputate at the thigh if the suppuration were very excessive, and acute suppuration of the knee-joint should occur. Should the disease affect the femur and run an unfavorable course, I should scarcely hope to save the patient by an operation so dangerous as amputation at the hip-joint. We may accomplish much by great care of the patients, who are generally youthful. A young girl with osteomyelitis and periostitis of the tibia had sixteen chills in twelve days, and nevertheless recovered, although part of the tibia became necrosed, and the foot was anchylosed.

I will here add a few remarks about suppurative periostitis of the third phalanx of the finger, which is, perhaps, the place where it most frequently occurs. As this inflammation in the hand and fingers is usually called panaritium, this periostitis of the last phalanx is termed *panaritium periostale.* This, like any periostitis, is very painful; it is a long while—sometimes eight or ten days—before the pus perforates outward. The termination in partial or total necrosis of the phalanx is common, and cannot be prevented even by an early incision, although we often have to make one to relieve the disagreeable, throbbing, burning pain, partly by the loss of blood, partly by splitting the periosteum. As the termination in suppuration can scarcely ever be avoided, we try to induce it by cataplasms, hand-baths, etc., and thus hasten the course.

Thus far we have only spoken of acute inflammation of the periosteum, and medulla of the long bones, but have not considered that of the *spongy bones.* Nor have we considered the question of inflammation of the bone-substance proper. Is there such a thing? I think this must be answered in the negative, for I consider that dilatation of the vessels, cell-infiltration, and serous imbibition of the tissue, in their various combinations, constitute the essence of acute inflammations. In the compact bone-substance (as in the cortical layer of a long bone) all these requirements cannot occur. In many places at least, the capillary vessels are so closely embedded in the Haversian canals that they cannot dilate much; a certain amount of serous infiltration of the bone is imaginable; but the firm bone-substance cannot possess much capability of swelling. If the term inflammation be made so general as to include every quantitative and qualitative disturbance of nutrition, it would be a very peculiar view, in which I do not participate. Every tissue attacked by inflammation changes its physical and chemical nature, and in acute inflammation of the soft parts this takes place rapidly; the connective tissue especially is quickly changed to a gelatinous, albuminous substance; the tissue of the cornea and cartilage may also change very quickly. For chemical reasons this is impossible in bone; time is required for the chalky salts of the bone to dissolve, and the bone-cartilage left deliquesces like other tissue. Hence, inflammation of compact bony tissue, severe though it be, cannot run its course very rapidly; it always takes a long while. The above refers only to compact bone-substance; *spongy* bones may readily become inflamed, that is, there may be inflammation of the medulla contained in the spongy bones which has the same peculiarities as that of the long bones, only it is not collected together as it is in them, but it is distributed in the meshes of the bones; each space contains many capillaries, connective tissue, fat-cells, and nerves; acute inflammation of the spongy bones first occurs in these interspaces, and gradually extends to the bone proper. What is called *acute ostitis* of a spongy bone is at first only acute osteomyelitis. This when idiopathic is rarely acute, but is usually chronic, sometimes subacute. On the other hand, there is a traumatic acute osteomyelitis of spongy bones, about which we shall here say something, although we have discussed its more important features when treating of suppuration of bone. Imagine an amputation wound close below the knee: the tibia has been sawed through its upper spongy part; traumatic inflammation occurs in the medulla of the bone, in the meshes of the bone-substance, with proliferation of vessels, cell-infiltration, etc.; this leads to development of granulations, which grow out from the medulla and soon form a granulating surface; this cicatrizes in the usual manner. But subsequently, if you have a chance to examine such a stump, you find that, at the sawed surface of the bone, the meshes are filled with bone-substance, and the outer layer of the spongy bone is transformed to compact bony substance; that is, the cicatrix in the bone has ossified. This is the normal termination not only of traumatic but of spontaneous ostitis the bony cicatrix ossifies. There may also be suppuration, putrefaction of the medulla of spongy bones, as in long bones; osteophlebitis and its consequences may also occur. In the lecture on suppuration of bone (p. 228) and healing of open fractures we treated fully of the changes which occur after the bone has lost its periosteum, of the development of granulations on the surface of compact bone-substance, and of the accompanying superficial necrosis.

Here I will merely add that we sometimes meet multiple inflammations of the bones as we do multiple acute inflammations of the soft parts (acute polyarticular rheumatism); these may occur simultaneously in the two corresponding bones of the lower extremities, or may follow each other; e. g., osteomyelitis of the tibia, suppurative inflammation of the knee-joint, osteomyelitis of the femur, purulent inflammation of the hip-joint; in one case there was also osteomyelitis of the other femur and purulent coxitis of the other side. Even such cases may possibly terminate favorably, but this is very rare; they usually end fatally.

My saying that I could not imagine an acute inflammation of *bony tissue* has caused some misunderstanding. In acute inflammation of bone no changes are observed in the (fully developed) osseous tissue, but only in the medulla and periosteum and their vessels. I do not underestimate the chemical changes (disturbances of nutrition) which go on in the tissues during inflammation; but we do not know them: we only conclude they occur from the changes we see in the tissues. We see that inflamed connective tissue swells, becomes cloudy, is infiltrated by wandering cells, softens, and finally breaks down into pus; and all this occurs within a few days. In bony tissue we see none of these changes; we do not see that it swells in acute inflammation, or that its interspaces (except the Haversian canals) fill with wandering cells; and we know that it does not suddenly break down into pus. We only know one *termination* of acute inflammation of bone, that is, death—necrosis; besides this, it may pass on into chronic inflammation. Hence we can only say it is probable that in acute inflammation of bone changes of nutrition occur, as in that of connective tissue; but there is, or rather from the nature of bone-tissue there can be, no morphological expression for this change.

[Osteomyelitis may occur spontaneously, or after "cold," or secondarily in the vicinity of acutely-inflamed joints, or as a result of severe crushing or subcutaneous fractures; also after acute infectious diseases, especially typhus, scarlatina, and measles, or after inflammations of any part of the body. But it is probable that neither catching cold nor injury is the sole actual cause, but that it is due rather to a general or local infection, like articular rheumatism and certain phlegmons, though we cannot always discover the source of the infection. After *Roser* and *Lücke* advanced the theory that this disease was of infectious origin, *Kocher*, *Rosenbach*, *Busch*, and others, showed experimentally that acute suppurative osteomyelitis could not be induced by chemical or mechanical injury, but resulted at once if putrefying matters were applied to fresh wounds of bone. The affection does not seem to be induced by any specific poison, but may be excited by a variety of putrid matters already in the body, or which may have entered through the alimentary canal, if an injury or irritation of the medulla of bone has been developed in any way. Rarely, several bones are attacked at the same time; we have a *multiple* osteomyelitis; we do not know if this is due to an infection acting at the same time on various points, or to passage of the poison from the point primarily affected. On examining the diseased bone, we find scattered in the medulla numerous punctate abscesses composed of pus-corpuscles and numerous micrococci, or larger collections of putrid pus containing also quantities of large multigranular cells, proliferating fatty tissue according to *Rosenbach*, and spindle-shaped elements. If infection has occurred in any way, there will be acute suppurations leading to decomposition of the medulla in the worst cases. The periosteum becomes diseased at once, or secondarily; either without direct connection with the primary abscess, or after it has opened. Often the neighboring joints are affected, either at the onset or later. There are also inflammations of the serous membranes, suppurative pleuritis and pericarditis, abscesses in the lungs, liver, etc. The disease proves fatal with typhoid symptoms and coma by general septic infection, or by pyæmia from absorption of pus, or lastly, after a protracted course from secondary inflammation of internal organs. Although between the severe rapidly fatal cases and the milder forms of osteomyelitis there is no essential etiological difference, those that are hopeless from the first are fortunately rare, and in some places never occur. Sometimes in the joint implicated there is development of gas, even before the abscess has opened; this is a bad sign. The pus in bones affected with osteomyelitis often contains fat-globules from the medulla and quantities of micrococci, which are also found in purulent thrombi of osseous veins, metastatic deposits in the kidneys, etc.

While acute osteomyelitis comes from infection of the blood-vessels of the bone by septic matters, there is another form of ostitis and osteomyelitis, probably also due to entrance of a foreign body into the vessels, but whose action is chiefly mechanical. This peculiar and etiologically very interesting form, described by *Englisch* and *Gussenbauer*, is ostitis of turners of mother-of-pearl; these workmen are mostly young, and live in an atmosphere impregnated with very fine powder of mother-of-pearl; they are frequently attacked by painful, multiple inflammations of the diaphyses of the long bones, with marked swelling of the periosteum, which have a subacute course, and according to present experience never lead to suppuration. The result of this osteoperiostitis is complete recovery; at most there is left some thickening of the periosteum, but relapses occur if patients resume their occupation. From chemical and microscopical examination of the mother-of-pearl dust, and clinical observations, *Gussenbauer* offers the following hypothesis as to the genesis of this disease: Besides inorganic, mother-of-pearl dust has organic constituents, which, being inhaled into the lungs, pass into the circulation; the molecules of the organic constituents are of such a size that they stick in the small arteries of the diaphyses, and there form emboli and induce inflammation of the bone and secondarily of the periosteum. In the diaphyses of the long bones there are minute arteries supplying a circumscribed territory (terminal arteries of *Cohnheim*). This explanation might account for the sudden painful occurrence of the disease, and the subsequent local swelling, as well as its being confined to the diaphysis, which is the only seat of the peculiar arrangement of the vessels.

A belief in the infectious nature of osteomyelitis will greatly influence the treatment. If there be fever, with morning remissions, give fifteen to twenty grains of quinine or ninety to one hundred and eighty grains of salicylate of soda daily during the remission. If certain of the diagnosis, the abscess should be opened early, with antiseptic precautions. An incision should be made down to the bone at the tender spot, and, if no pus be found under the periosteum, an opening should be made in the bone with a trephine or chisel; the pus should be washed out, the wound disinfected, and an antiseptic dressing applied. If, instead of being circumscribed, the pus be infiltrated through the bone, opening the medullary cavity will probably do little good.

The treatment of ordinary subacute periostitis is more simple and easy; we may quietly await suppuration and incise when fluctuation is evident; till then use moist-warmth, rest, elevation, and moderate compression of the limb. When pus appears at any point, open with antiseptic precautions, drain, and apply *Lister's* dressing till the cavity fills up. Fixation of a limb affected with acute osteomyelitis is the more important the more acute and painful the process, and it is particularly necessary when there is detachment of an epiphysis. In such cases plaster dressings were formerly applied, but for *Lister's* dressing splints and starched gauze (organtin) are better, or else permanent extension and gutta-percha splints.

Under suitable treatment, after evacuation of the pus, fever is arrested, swelling disappears from the limb, and the disease passes into a chronic stage. During this time moderate discharge of pus continues, and gradually the necrosed portion of bone becomes detached from the healthy part. This constitutes the process of repair and restitution: on the one hand, the sequestrum is eliminated; on the other hand, the loss of substance in the bone is replaced. Suppurative inflammation of joints requires special treatment, of which we will treat in the next chapter.]

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We now come to *acute inflammation of the joints.* As we have previously spoken of traumatic articular inflammations, you already know some of the peculiarities of diseased joints. You also know that serous membranes have a great tendency to excrete fluid exudation when irritated, but that this exudation may also contain pus, if the inflammatory irritation be very intense. As there is a pleurisy with effusion of sero-fibrinous fluid (the ordinary form), and a variety with purulent effusion (so-called empyema), so in joints we speak of serous synovitis, or hydrops, and of purulent synovitis, or empyema; both forms of the disease may be either acute or chronic, and they induce various diseases of the cartilage, bone, articular capsule, periosteum, and surrounding muscles. You will see that it is always more complicated with these diseases the more complicated the affected part is. Of late, great importance has been attached (especially by French surgeons) to speaking, first, of diseases of the synovial membrane, then of those of the cartilage, articular capsule, and bone, corresponding to the anatomical conditions. Correct as this division would be, if it were only a question of representing the pathological anatomical changes, it is of little use in practice. The surgeon always views inflammation of the joint as a whole, and, although he should know which part of the joint suffers most, this is only a part of what he should know; course, symptoms, and constitutional state, equally demand his attention, and determine the treatment. Hence the entire clinical appearance will determine the divisions of this, as of many other diseases.

At present we are speaking only of apparently spontaneous acute inflammations of the joints. In many cases they are evidently due to catching cold, in other cases their causes are obscure. Some of the more subacute cases are of metastatic nature and appear as pyæmia. But at present we shall speak only of the idiopathic inflammations, which, in contradistinction to the traumatic, are termed *rheumatic*, as they are often due to cold. Patients requiring your aid for such acute inflammations of the joints, will present somewhat different symptoms. If, for illustration, we again take the knee-joint, you will have about the following picture: A strong, otherwise healthy man has taken to bed, because for a day or two his knee has been swollen, hot, and painful; you find this on examining the knee, you also find distinct fluctuation in the joint, and that the patella is somewhat lifted up, and always rises again if pressed down; the skin over the joint is not red; the patient lies with his leg stretched out in bed, has no fever, and, if you ask him, can bend and extend the knee, though with some difficulty. You here have an *acute serous synovitis*, or *hydrops genu acutus*. The anatomical condition of the knee is as follows: the synovial membrane is slightly swollen and moderately vascular; the articular cavity full of serum, which has mingled with the synovia; there are a few flocculi of fibrine in the fluid, the rest of the joint is healthy. Anatomically the state is just like a subacute bursitis tendinum or a moderate pleurisy. This disease is generally cured without difficulty; quiet, repeatedly painting with tincture of iodine, or a few blisters, or compression with wet bandages, suffice to remove the affection in a few days, or at least to take off its acuteness; all the symptoms of the acute inflammation may subside, the patient may go about with scarcely any difficulty, but there remains too much fluid in the joint, a hydrops chronicus of the joint is left.

You may be called to another patient with inflammation of the knee-joint. A few days previously the young man has caught cold; soon after this his knee has begun to pain, high fever has come on, perhaps a heavy chill; the joint has constantly grown more painful. The patient lies in bed, with the knee flexed so that the thigh is strongly rotated outward and abducted; he resists every attempt to move the leg, as it causes him terrible pain. The knee-joint is greatly swollen and feels hot, but there is no fluctuation, the skin is œdematous and red about the knee, the whole leg also is œdematous; on account of the pain it is impossible to extend the knee or to flex it more. What a contrast to the former case! If you have a chance to examine the joint in this stage, you find great swelling of the synovial membrane; it is very red, puffy, and microscopically appears infiltrated with plastic matter and serum. In the joint there is usually a little flocculent pus mixed with the synovia, there may also be pure pus. The surface of the cartilage looks cloudy, and microscopically perhaps shows little change beyond turbidity of the hyaline substance; possibly the cartilage cavities are somewhat enlarged and filled with an unusual number of cells. The tissue of the articular capsule is œdematous. Here you have a *purulent very acute synovitis*, in which the cartilage threatens to participate; should the disease continue, and the pus in the joint increase, you may correctly call it *empyema of the joint.*

The difference between the first and second forms of acute synovitis is essentially that, in the second, the tissue of the synovial membrane is deeply affected, while in the first the increased secretion is the chief feature. Between these two forms are subacute cases, in which the secretion becomes purulent and collects in great quantity, without there being any great destruction of the synovial membrane. *R. Volkmann* calls this "catarrhal inflammation" of the joint; it is somewhat more painful than ordinary acute hydrops, from which the catarrhal purulent form may proceed, though this is rarely the case. I have already said what was necessary about the course and treatment of acute hydrops. The course and results of the more parenchymatous synovitis, which is predisposed to suppuration, depend greatly on when the treatment is begun and what it is. Usually a few leeches are applied and then the joint is poulticed, from an idea of the old school, that rheumatic articular inflammations should be treated with warm applications. I consider leeches almost useless in these affections; perhaps there may be a question about keeping the limb warm, for this is often pleasant to the patient; it alleviates the pain in inflammations of the serous membranes, often more so than cold does; at least the latter must act for some time before having a favorable effect. I explain this as follows: The warm applications induce fluxion to the vessels of the skin, and thus empty those of the synovial membrane; but this effect is not long continued; fluxion to the inflamed deeper parts returns again, and is stronger than to the artificially-warmed skin. On application of a large bladder of ice to the joint, the vessels of the skin contract, and perhaps drive the blood to the vessels of the inflamed part more strongly than before, till gradually the cold has its effect on these also, and if the cold continues the effect becomes permanent. It seems more rational always to use cold in these cases; in very acute inflammations of the joint the employment of ice-bladders has also proved very practical. Besides using cold, you may also induce active derivation to the skin by strong tincture of iodine, or by a large blister. But besides these remedies it is most important to bring the joint into a proper position and keep it there, for, if we do not obtain a perfect cure, and the joint remains stiff, the flexed position of the knee, which is so frequent, is a very unfortunate addition to the stiffness, as it renders the limb nearly if not entirely useless.

The malposition should be relieved; this should be done for each joint in such a way that in case of complete stiffness its position shall be most favorable. The hip and knee-joint should be extended, the foot and elbow at right angles; the wrist and shoulder do not get out of position; the former usually remains extended, the latter usually takes such a position that the arm lies against the thorax. There is very great difference in the frequency of acute disease in the different joints; the knee is most frequently affected, then the elbow and wrist; acute inflammation of the hip, shoulder, and ankle, is rare. Acute articular inflammations are more frequent in young persons than in old, but hardly ever occur in children. But, to return again to the improvement of the position of the joint: you will tell me this is impossible. Chloroform is here useful; this remedy has become most important in the treatment of inflammations of the joints. You narcotize the patient deeply, and can then move the limb without trouble; the muscles, which previously contracted on the least touch, now yield without difficulty. If we continue with our former hypothetical case, you extend the knee, envelop it in a thick layer of wadding, and apply a plaster-splint from the foot to the middle of the thigh. When the patient awakes, he will at first complain of severe pain; give him a quarter of a grain of morphia and apply one or two bladders of ice over the plaster-splint to the knee; the cold acts slowly, but finally proves effective, and in twenty-four hours the patient feels tolerably comfortable. The slight compression made by the well-padded plaster-splint also has a favorable antiphlogistic action; if there be fever, you may give cooling medicines and saline purgatives; but the patient needs no further treatment. Before applying the dressing, you may have the limb rubbed with mercurial ointment or painted with tincture of iodine. It is best to apply the dressing even in the most acute stage; of course it must be done very carefully, avoiding any strangulating pressure. Recently it has been shown that, even in very acute inflammations of the joints, surprising results may be obtained by extension with weights. It is very interesting to observe how a continued moderate traction lessens the pain in the joint and relaxes the muscles. But much depends on the application of the dressings, and I cannot too strongly urge on you to attend carefully to these apparently simple mechanical things, whose importance you will not correctly estimate till thrown on your own resources in practice, and obliged to attend to the minutest details yourself.

[The treatment in acute non-traumatic suppuration of joints has changed greatly within a few years. If, after fixation of the joint, the inflammation threatens progress, we may open the joint freely with antiseptic precautions, as was described when treating of traumatic cases. Pus is evacuated and all recesses of the synovial sac are to be washed out with a two-per-cent. solution of carbolic acid; the joint should be drained, disinfected with a five-per-cent. solution of chloride of zinc, and a *Lister's* dressing applied; then the limb kept in proper splints. Instead of *Lister's* dressing, permanent irrigation with *Burow's* solution may be indicated; in patients with high fever this has the advantage of being an excellent means for reducing temperature. By this treatment we may succeed in curing the case with a movable joint if the articular cartilages have not been destroyed; and at any rate we may prevent periarticular abscesses and absorption of pus. If extensive suppuration of the soft parts has begun, permanent irrigation is particularly indicated, but will often prove ineffectual, and amputation will be necessary.]

If called to the case early, you may sometimes not only arrest the acute stage of the disease, but may preserve to your patient a movable joint. But, even if called late, the above treatment should be pursued. If the pain is relieved and the fever ceases, you may remove the dressing in a few weeks, for the disease lasts several weeks under any circumstances; perhaps three to five months may elapse before the inflammation entirely disappears; gradually the normal condition and the former mobility return, then the patient should be earnestly warned against taking cold or excessive motion, for a second attack might not turn out so well.

Supposing the acute process does not subside under the treatment instituted, but continues to progress, it may pass into a chronic form, or remain acute; we shall hereafter treat of the former case. Let us at present suppose that the pain, instead of subsiding, becomes more severe, and you are obliged to split the dressing along the front; you find the knee more swollen, distinctly fluctuating, and the patella very movable, while the patient has high fever. If the disease continues, the fluctuation may extend farther and farther, upward to the thigh, for instance, and the subcutaneous cellular tissue of the thigh and leg may participate in the suppuration. Formerly this extension was attributed to subcutaneous bursting, or partial suppuration of the synovial sacs around the joint, especially of the large one under the tendon of the quadriceps femoris, and of the bursa poplitea; to prevent this misfortune it was considered advisable to tap the joint with a trocar, in the above stage of the disease, to let out most of the pus, and then carefully close the opening. From my own experience I should consider this operation as rarely indicated, for I have convinced myself, by careful examinations of patients, and occasionally of the cadaver, that these periarticular abscesses in the cellular tissue, occurring in acute synovitis, and also in ostitis of the articular extremities, form separately, and break into the joint late, if they do so at all. With the development of these abscesses the general condition of the patient is usually impaired; he has high fever, with intercurrent chills, his cheeks fall in, he emaciates, loses his appetite, and becomes sleepless. Quinine and opium finally lose their effect, and, unless you amputate the thigh early enough, the patient dies from the exhausting suppuration and continued fever; perhaps, also, he may have metastatic abscesses. If, by the applications of ice, by one or more incisions for evacuating the pus, by quinine and opium, you succeed in breaking the acute stage of the disease, and making it chronic, you will not obtain a movable joint, but even if it is flexed at a right angle, the leg will be useful; this is the best result that we can gain after days and weeks of anxiety and care, if the inflammation reaches the above grade. The anatomical changes in a knee-joint in this stage of inflammation are as follows: The joint is filled with thick yellow pus, mixed with fibrinous flocculi; the synovial membrane is covered with dense purulent fibrous rinds, under which it is very red and puffy, partly ulcerated; the cartilage is partly broken down into pulp, partly necrosed and peels off; the bone under it is very red or infiltrated (osteomyelitis; usually in these cases a secondary, rarely a primary disease).

The prognosis of this disease is not very bad in young, vigorous persons, when the proper treatment is resorted to early; it is very bad, almost absolutely fatal, in old, decrepit persons.

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In the above I have pictured to you typical cases of the two forms of synovitis, the serous and parenchymatous (purulent), and am satisfied that in practice you will readily recognize these pictures again; and you will have no difficulty in applying what has been said of the knee to other joints. Now I must add that there is still another acute or subacute form of articular inflammation, which offers some peculiarities. I refer to *acute articular rheumatism.* This very peculiar disease, which will be treated of more fully in the lectures on internal medicine, is characterized by its attacking several joints at once, and its tendency to cause inflammations of other serous membranes, such as the pericardium and endocardium, the pleura, and rarely the peritonæum and arachnoid. This simultaneous disease of these membranes and of the joints marks the affection as one implicating the whole body from the start; indeed, from the importance of the organ affected, the pericarditis and endocarditis are often so prominent, and so much influence the treatment, that the surgical treatment of the joints is a very secondary matter; this is the more apt to be the case, as this disease, although very painful, rarely proves dangerous to the limb or to life. The chief symptoms of the local affection, beyond which the disease rarely proceeds, are, great pain in the joint on every motion or touch, œdema of the surrounding soft parts, and rarely redness of the skin. From the few autopsies that have been made, it appears that the synovia increases somewhat, is sometimes mixed with flocculi of pus, and the synovial membrane is swollen and red; the cartilage is seldom implicated; the collection of fluid is not often so great as to cause fluctuation. Acute rheumatism is very frequent, but it is rarely fatal, so that the pathological anatomical appearances are little known. From all the symptoms of this disease, it is evidently a specific, limited disease, of a peculiar character, but with a course so atypical, and causes so obscure, that its actual character has not yet been determined. I have my doubts whether, besides this *polyarticular*, we can speak of a *monarticular acute rheumatism*, for it is just the multiplicity of the points of inflammation, and their slight tendency to suppurate, that characterize the disease; at all events, I should not consider an inflammation limited to one joint as a symptom of acute rheumatism, unless pleurisy, pericarditis, or some other complication peculiar to rheumatism, also occurred; should none of these come on, the disease is purely local, a simple inflammation of the joint, which is probably called rheumatic simply because it is supposed to be due to catching cold. In acute rheumatism, the resolution of the articular inflammation and the restoration of the joint to its functions are so common that we rarely see any other termination. That the disease is tedious, and generally lasts six or eight weeks, is not so much due to the duration of the affection in a single joint as to its attacking first one joint, then another, and exacerbations readily occurring in joints that had recovered; thus the disease proves tedious, both for physician and patient, and the greatest watchfulness and care are necessary to avoid all sources of injury that may again arouse the disease. It is exceedingly rare for one of the affected joints to go on to intense suppuration or empyema; more frequently, in spite of the subsidence of the disease, a joint remains stiff and painful, and passes into a state of chronic inflammation. You see that the prognosis of this disease, as far as it concerns the joint, may be called very favorable; without any interference from the physician, the joint-inflammations generally run a favorable course. Hence all that we do for the local disease is to envelop the joint in wadding, tow, oakum, or wool, to protect it from changes of temperature. Mild cutaneous irritants and painting with tincture of iodine may also be useful. For alleviating the pain in the joints and hastening the course of the disease, *Stromeyer* and others recommend the employment of bladders of ice, and generally keeping the joint cool, rather than warm. But I think this treatment will find few disciples, for it is quite troublesome, and experience shows that the articular inflammations get on well without such applications. Internally, we may give diuretics, diaphoretics, or cooling salts; in heart-affections, local antiphlogistics, digitalis, etc., are indicated, as will be taught you more particularly in special pathologies, and in the medical clinics.

Next to acute rheumatism comes acute *arthritic* inflammation of the joints. The attack of podagra or chiragra is also specific and belongs to true gout; here, also, the articular inflammation is an acute serous synovitis, but with very little secretion of fluid in the joint. But one thing peculiar to acute arthritic inflammation is the never-failing coincident inflammation of the surrounding parts: the periosteum, sheaths of the tendons, but especially of the skin; this always reddens, becomes glistening and tense, as in erysipelas, and is very painful; it even desquamates occasionally after the attack. Acute arthritic articular inflammation is far more painful than rheumatic. We shall hereafter speak of the treatment of arthritis and the arthritic diathesis.

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There is still another variety of acute articular inflammation, the *metastatic*, about which we shall have something more to say when treating of pyæmia. Acute or subacute metastatic inflammation of the joint is usually at first serous, but soon purely suppurative synovitis. Several forms may be distinguished:

1. *Gonorrhœal inflammation of the joints.* This occurs in men suffering from gonorrhœa; occasionally, also, it occurs after the introduction of bougies into the urethra; it attacks the knee-joint almost exclusively. Some authors assert that it is especially apt to develop when the gonorrhœa is arrested suddenly. This is not my own experience. In proportion to the frequency of gonorrhœa, it is very rare but I have seen it quite frequently when a patient with active

gonorrhœa has caught cold. The incomprehensible connection between purulent catarrh of the urethra and inflammations of the knee-joint might be denied, and the simultaneous occurrence of the two diseases be considered as accidental; but the experience of too many surgeons, and also cases where inflammations of the knee-joint occur after other irritations of the urethra (as by bougies), speak in its favor. Gonorrhœal gonarthritis usually attacks both sides, and is a subacute serous synovitis, which generally soon disappears under proper rest, avoidance of new irritation of the urethra, blisters, tincture of iodine, and slight compression of the joint; and, after reabsorption of the fluid, it ends in perfect cure. But irritability of the joint is apt to remain, and not unfrequently the same person getting another gonorrhœa is again attacked with inflammation of the joints. In some cases chronic articular rheumatism is said to follow gonorrhœal gonarthritis.

2. *Pyæmic inflammation* also occurs very frequently in one knee, as well as in the ankle, shoulder, elbow, and wrist; rarely in the hip. It is a pure purulent synovitis, subsequently accompanied by suppuration of the periarticular cellular tissue, but usually with subacute course, and hence we do not always find it fully developed at the time of autopsy. Pyæmic patients do not always die with suppuration of the joint, and I have witnessed reabsorption in cases where the patient lived through the purulent infection. The treatment does not differ from that above given; if the collection of pus is excessive, puncture will relieve the pain. Suppurations of the joint due to injuries, and lacerations of the urethra by careless catheterization, and usually accompanied by chills, are of course pyæmic, not gonorrhœal. In Berlin I treated a young man who had a rupture of the urethra caused by bougies, and consequently an abscess of the left shoulder, with suppuration of the acromial joint of the clavicle, which induced subluxation of that bone. The patient recovered perfectly; and, as the abscess was not large, it was not opened. A year later I saw the young man again. The abscess had become somewhat smaller, fluctuation was still distinct; but, as it caused no disturbance of function or other difficulty, and the patient was blooming and healthy, I avoided opening the abscess, and advise you to do the same with cold abscesses which evidently communicate with a joint, as the opening does little good and may do much harm, by possibly inducing acute inflammation of the joint and very disagreeable results.

3. *Puerperal inflammations of the joints.* Puerperal fever is a form of pyæmia that may occur after parturition. Hence, the suppurative inflammations of the joints occurring at that time come under

the above category of pyæmic, suppurative synovitis. But not unfrequently, the third or fourth week after parturition, there is an acute suppurative inflammation of the knee and elbow joints, which has been referred to various causes. Some say it is a simple form of acute articular inflammation due to catching cold, to which women are particularly liable after confinement, because they perspire so much. Others are of the opinion that these late inflammations of the joints are also symptoms of pyæmia that have been overlooked and are isolated, and hence consider them as metastatic. Let this be as it may, it is at all events certain that these cases have nothing specific. They run either an acute or subacute course, and, under suitable treatment, may be so controlled that the joint will remain movable; but sometimes a more chronic course begins later and terminates in anchylosis. The prognosis is not very bad. They rarely reach the highest grade of acuteness. The treatment is the same as that already given for acute suppurative synovitis.

I would also mention that purulent articular inflammations occur in the pyæmia of the newly-born; children are even occasionally born with them, as has been witnessed by myself and others. Inflammations of the joints may develop and even run their course during fœtal life, as is shown by the cases where children are born with joints fully developed but anchylosed.

## APPENDIX TO CHAPTERS I.-XI.

### *RETROSPECT.—GENERAL REMARKS ON ACUTE INFLAMMATION.*

GENTLEMEN: Thus far I have given you a number of clinical surgical pictures representing various forms of acute inflammation. We have seen injuries and their results, as well as the acute surgical diseases occurring without injury, and have studied the disturbed physiological processes, the means of their removal, and the process of this removal. It seemed as if this method would be stimulating for you, and that it was permissible, as you were supposed to have some knowledge of general pathology and some starting-point for pathological, physiological, and histological investigations. Still, it will not be superfluous, at the close of this first and most extensive section of our work, to give a brief *résumé* of the present views of inflammation, which have been greatly advanced by recent labors of *Cohnheim*, *Samuel*, *Arnold*, and others.

I will begin by saying that from our ignorance of the participation of nerves in inflammation, we must leave them out of the question. Vessels, blood, and tissue form almost exclusively the objects of our study.

Dilatation of the blood-vessels is an important factor in inflammation; still, neither the hyperæmia from hinderance to the current of blood in the veins (congestive hyperæmia) or dilatation of the arteries from paralysis of their walls (as in the rabbit's ear after division of the cervical sympathetic), nor the sudden primary dilatation from mechanical and chemical irritations, necessarily leads to inflammation. About the latter form of vascular dilatation I have something to add to what has already been said. It is about the following symptom: You rub the eye, and it becomes red; you rub the skin, and it becomes red, as it also does if you apply warm water; you put snow on the skin, and it becomes white, then red. All of these reddenings soon pass off if their causes only acted a short time and were soon removed. The investigations mentioned in Lecture V. referred to the mode of origin of these hyperæmias, but they are now considered unsatisfactory. The symptom itself is completely estimated by *Cohnheim*; still, even under the action of heat, cold, and chemical influences, if we suppose a direct momentary paralysis of the vascular walls, from what we have thus far seen, it appears strange that a paralyzing influence should extend from a circumscribed pressure or tear to an extensive portion of surrounding vascular territory, with a sort of wave-like motion. It seems to me we know no more about this "affluxus" to the "stimulus" than we formerly did. But it is important, as *Cohnheim* has shown that where inflammations occur after physical or chemical influences, these primary fluxions may have passed over long before the new hyperæmia which leads to and continues with the inflammation; and the primary fluxions may entirely fail, but a regular inflammation with its hyperæmia nevertheless occurs. Hence the fluxion immediately following the irritation is not an absolutely necessary factor of the inflammation.

A rabbit's ear whose vessels have been paralyzed and dilated by section of the sympathetic does not inflame; its tissue becomes more tense from œdema, but nothing more; there is no further disturbance of nutrition in the vessels and tissues.

Extensive congestion, however, is more serious. It has already been stated in Lecture V. that slight increase of intravascular pressure, such as occurs after moderate injuries, quickly passes over and has no effect on the inflammation. But if the congestion be very extensive and cannot be equalized, there is so copious an exudation of serum in the tissue (œdema) that it cannot be carried off by the lymphatics; sometimes there is free escape of red blood-corpuscles through the walls of the capillaries into the tissues (diapedesis). *Cohnheim* stated it as probable that the diapedesis resulted through openings in the capillary walls. *Arnold* not only confirmed this, but indicated the so-called stigmata (the small openings which become visible between the cells forming the capillaries, after staining with silver) as the point of escape, and also showed that blood-serum flowed out through these stigmata. If the hinderance to the circulation be of such a nature that blood can continue to flow, the only results will be œdema and diapedesis; if the circulation be entirely arrested, gangrene results.

Coming at last to hyperæmia as it occurs in inflammation, it is neither the immediate result of temporary inflammation, nor of paralysis of the vaso-motor nerves, nor of obstruction to the circulation, but of a peculiar alteration of the walls of the vessels, especially of the capillaries and veins. What chemical or physical changes occur at the same time in the walls of the vessels cannot be stated; but we conclude that the vessels in the inflamed part are permanently dilated, and permit the free escape of white blood-corpuscles (not only at the stigmata, but at any point in their walls), and that the substance of these vessels is softened and more yielding. Why this is so, certainly cannot be determined in all cases; it is considered as a direct effect of the cause of the inflammation, though it does not occur for some hours. The inflamed borders and areolæ around sharply-bordered cuts or stabs are just as difficult to explain as the primary fluxions. We must even involuntarily suppose that a disturbance can never be accurately confined to the part supplied by a certain vessel, but that it must spread somewhat, least so in cuts, stabs, or rapid burns, and most so after certain chemical actions. Still, this is no true explanation; it is merely a limitation of observation.

Let us now consider the blood and its circulation in inflamed tissue. Primary fluxion is accompanied by greatly increased activity of the movement of the blood, especially in the arteries, which again becomes normal as the primary dilatation of the vessels recedes. In the vessels which dilate permanently, in the borders and areola of the inflammation, the rapidity of the circulation gradually diminishes, especially in the veins; the blood may move by impulses, or occasionally stop completely. This stasis, which is not at once accompanied by coagulation of the blood, was formerly regarded as a necessary part of true inflammation, and had many explanations, which hardly interest us now, as we know that many inflammations run their course without stasis, as well as that this stasis often disappears in spite of progressing inflammation. If it continues, the blood finally coagulates in the vessel (thrombosis), the results of which vary with local conditions and the extent; there may be a return to the normal state by collateral dilatation, or gangrene may result. The circulation in the inflamed part is at first slow and irregular, and again becomes normal. Meantime numerous white blood-cells collect along the walls of the small veins and capillaries; then they wander through the walls of the vessels into the tissues, whose interstices become filled (cellular, or, if excessive, purulent infiltration); and finally they reach the surface (superficial suppuration, purulent catarrh).

We have now the complete picture of acute inflammation; but the process may recede at the time of dilatation of the vessels and arrest of the white blood-cells, and even subsequently, when cellular infiltration has advanced quite far, without leaving any change perceptible in the tissue that has been infiltrated or the vessels which have been dilated. But at a certain height of the purulent infiltration the tissue disappears entirely, and is replaced by pus (an abscess forms), or by an interstitial neoplasia (granulation tissue), which, if it does not die, becomes connective tissue (cicatrix), with vessels and nerves.

The question arises, What causes this atrophy of inflamed tissue? Is it the direct effect of the cause of the inflammation or of the cellular infiltration? Here we come to the third important point in inflammation, namely, the part taken by the *tissue* itself. If we first consider the inflammations caused by known chemical or physical causes, it is evident they cannot act on the vessels and blood without at the same time affecting the tissue. *Samuel* starts from the inflammation induced by chemical means, and explains it as a resultant of the union of the cause of the inflammation with the tissue, the walls of the blood-vessels and the blood. The wandering of the blood-cells, their infiltration in the tissue, and the accompanying changes, he regards as secondary processes. If the action of concentrated sulphuric acid on the tissue causes such a metamorphosis that circulation of blood and other fluids is no longer possible, the tissue is directly killed; but the most essential thing in the inflammation is the change of tissue affected by dilute sulphuric acid (whether at the borders of a part cauterized by strong acid or where only dilute acid was used), where circulation still continues. According to this, if I have rightly understood *Samuel's* explanation, the disturbance in the inflamed tissue would vary in different cases, according as the active cause was an acid, an alkali, ethereal oil (as oil of turpentine), or an acrid oil (as croton-oil), etc. The condition of the inflamed tissue would differ also with action of extreme cold, great heat, crushing, after steam on exposed surfaces or serous membranes, etc. So we should have to renounce entirely a uniform representation of the chemical processes in the inflamed tissue. I do not know if this view will ever prove popular in this form. Hitherto we have classed these changes of tissue at the seat of inflammation all together; just as by concussion of the brain we should mean not only the moment of concussion, but also its immediate effect on the brain and its functions. If the concussion be followed by inflammation of the brain, the changes caused by the concussion may influence the nature and extent of the inflammation; but we do not say that a brain suffering from concussion is already inflamed. The same is true of contusions: if the normal function of a tissue has been affected by a concussion, but its function not entirely destroyed, the circulation will differ from normal, and this modification we call inflammation, but do not so term the immediate result of the contusion. The processes in the tissues, after chemical, physical, or mechanical injuries, are essentially similar, differing only in extent and intensity; they are what we term inflammation, and in it the tissue itself plays an important part, which varies with the way the cause has directly affected the tissue.

A constant perceptible result of acute inflammation is dilatation of the veins and capillaries, with escape of white blood-cells and certain disturbances of the physiological functions of the affected tissue. For all this to occur, one function of the vessels, especially that of the cellular elements of keeping the blood in the channels formed by them, must be disturbed; but would such a disturbance be confined to the walls of the vessels, and not extend to the adjacent tissue? This is not very probable. The granular cloudiness occurring in inflamed muscle, the indistinctness of the filaments in inflamed connective tissue, the granular disintegration in inflamed nerve-filaments, the rapid loss of color of red blood-cells in acutely inflamed tissue, all indicate that certain constant changes go on in the tissue also, which usually lead to gradual solution or death of the tissues, unless gangrene occurs from rapid increase of the process. I acknowledge there is no proof that these changes begin *simultaneously* with those in the vessels, and that they may be regarded as an immediate result of the latter; for if we find these alterations of tissue without dilatation of the vessels and cell-emigration, or if we artificially cause this state by obstructing the circulation to the injured part (*Samuel*), there may be a doubt as to whether it is to be termed inflammation in the ordinary sense (*Cohnheim*). But, on the other hand, attempts have been made to distinguish the changed condition of the vessels which permits the extensive escape of white blood-cells from inflammation. When studying chronic inflammation, we shall see that all of these factors can occur separately, and that it is only their combination which forms what we call inflammation.

*Virchow* located the inflammatory disturbances chiefly in the tissue; he was led to this partly by the microscopic changes just mentioned, partly from the observation that on irritation young cells appeared even in non-vascular tissues, like the cornea and cartilage, just as they do in vascular tissues. These latter observations, which were made at a time when the emigration of white blood-cells was not understood, can now be differently interpreted (Lecture VI.). We doubt now just as little as formerly that cartilage-cells and some others, as certain endothelia of serous membranes (*Rindfleisch*, *Kundrat*), young epithelial cells (*Remak*, *Buhl*, *Rindfleisch*), etc., on being irritated in a certain way, will form new protoplasm and new cells in themselves, will divide up, and may thus lead to formation of new tissue. It is still doubtful whether all cells thus formed have independent movements, like pus-cells; but very few observers now believe that developed connective tissue, corneal or bone corpuscles, acquire this peculiarity; it is pretty generally recognized that formation of pus does not result from local proliferation of fixed connective-tissue cells, according to *Virchow's* theory. Many regard it as still undecided how much the wandering cells have to do with inflammatory new formations; from my observations I can hardly doubt that the tissue which causes healing by first intention, as well as granulation tissue, may proceed from wandering cells, although another mode is possible (by offshoots, direct outgrowth from the tissue, Lecture VI.). The transformation of wandering cells into connective tissue seems to me quite plausible, for, according to my investigations, they probably originated from connective-tissue cells, namely, from the stellate cells, filaments of lymphatic glands. Of late, attempts have been made to explain *why* the above-mentioned tissue-cells, such as cartilage-cells, after certain irritations, begin to enlarge, divide, and finally to produce new tissue, by the hypothesis that every protoplasm, supplied with proper nourishment, would grow and divide up if not hindered by the pressure of the tissue in which it develops; the partial escape of the nucleus, as from injury or increased distensibility of the tissue, the nutritive conditions being otherwise good, is said to be enough to start the remains of the cell into growth. This hypothesis, which was advanced by *Thiersch* for another object, and which has been warmly taken up and generalized by *Samuel*, seems very ingenious, and I think it may prove the fruitful basis of future observations. But tissue-development is dependent on other important factors besides the conditions of nutrition and pressure, as on inherited peculiarities of the protoplasm; and the above hypothesis does not suit all cases—for instance, the endogenous cell-development of the endothelium after inflammatory irritation of the peritoneum.

It is not known whether there is a primary disturbance of nutrition in the tissues themselves, independent of the blood-vessels and their functions, that induces the specific inflammatory alteration in the vessels. The deposit of urates in the tissue of certain parts of the body in arthritis is usually regarded as of this nature; but the deposit requires participation of the vessels, and so they and the tissues are simultaneously affected. An experiment of *Cohnheim* shows that continued exclusion of blood from a blood-vessel may so affect its walls that when the blood again enters there will be a free emigration of white blood-cells. It was mentioned above that continued stasis of the blood did not have this effect on the walls of the vessel where it was stagnated; but from clinical grounds it is probable that the pressure of extensively and rapidly distended vessels on the parts around has something to do with their inflammation.

It is very probable that inflammations may be induced not only by chemical, physical, and mechanical causes, which act from without directly on certain parts of the body, but also by primary disturbances of nutrition in the tissues and of the circulation, which develop in the body without perceptible cause.

I must not forget to mention one symptom which formerly played a great role in inflammation, but is now hardly mentioned; that is, the *formation of fibrine* in some inflammations. This occurs chiefly, indeed almost exclusively, in inflammation of the connective tissue, and sometimes on the surface of serous sacs, of fresh and granulating wounds, and of mucous membranes (of pharynx, larynx, and bronchi); in other cases the nutrient fluid in the connective tissue assumes a fibrinous rigidity. It has been already mentioned that the formation of fibrine is not from an excess of fibrine in the blood, but from chemical alteration in the inflamed parts. Fibrine forms in the inflamed tissue, but is not a constant result of inflammation. The great difference of the other symptoms occurring with fibrinous inflammations is remarkable. While rapid formation of a moderate amount of fibrine favors healing by the first intention and partial adhesion of the surfaces of serous membranes, when often scarcely a trace of inflammation or fever is perceptible, in other cases, from some enigmatical cause, a very moderate fibrinous deposit in the tissues (as fibrinous deposit on the mucous membrane of the throat diphtheria) causes death. It is very evident that fibrinous hardening of the tissue fluids is one of the severest alterations of their nutrition; and, as experience shows, it often ends in necrosis. Still, the severe general symptoms and extensive inflammatory redness in these processes cannot be due simply to the formation of fibrine, but seem referable to absorption of the products of decomposition in the diseased tissue, which has a very rapid poisonous action. In the acute inflammations with formation of fibrine there seems to be a scale of malignancy similar to those without such formation, so that this would seem to be rather an accident due to the variety of the tissue and its locality; and while its significance is very important, it is not essential to the inflammation, nor does it materially change the course.

The *serous transudation* also, which accompanies acute inflammations, deserves a short notice. In many cases it certainly is the result of change of pressure in the vessels at the seat of inflammation; but it is just as much due to impaired function of the walls of the vessels and of the tissue; it is often a prominent symptom in inflammations of the connective tissue, especially of serous membranes. The walls of the vessels cannot hold the serum of the blood; the tissue does not prepare it; veins and lymphatics do not carry it away, especially if they are covered and stopped up by fibrine (in inflammation of serous surfaces on which the lymphatics open). The serum in acutely inflamed tissue is essentially different from that which, without inflammation, causes dropsy, for it not only contains wandering cells and disintegrated red blood-cells, but also the soluble products of the inflammation. The removal of this fluid by the veins and lymphatics releases the tissues from a considerable pressure and carries off the injurious products, it is true; but part of it at least is carried into the blood, and probably causes the inflammatory fever. This has already been fully treated of.

Now we might speak of the causes why circumscribed and often purely mechanical irritations, acting on small portions of the body, occasionally excite such intense spreading inflammations, and of the way these spread. But I will not now trouble you further with this subject. I have already said something about it in Lecture XXI., and shall hereafter have occasion to say more.

Pathological anatomists have paid too little attention to these questions; surgeons see their importance too often, and seek in vain for a means to arrest these spreading inflammations. In the clinic there will be many opportunities to call your attention to these important points.

It is in the nature of our times to undervalue the significance and practical value of these so-called theoretical reflections with which I have perhaps fatigued some of you. But hereafter, when you have been in practice for some years, you will hardly be able to read and understand a medical work if, during your student-life, you have not acquired a basis on which to build. After some years of practice some of you, who are now sated with lectures, will long to hear a continuous scientific exposition of important morbid processes.

# CHAPTER XII.

## GANGRENE.

### LECTURE XXIII.

Dry, Moist Gangrene.—Immediate Causes.—Process of Detachment.—Varieties of Gangrene according to the Remote Causes.—1. Loss of Vitality of the Tissue from Mechanical or Chemical Causes.—2. Complete Arrest of the Afflux and Efflux of Blood.—Incarceration.—Continued Pressure.—Decubitus.—Great Tension of the Tissue.—3. Complete Arrest of the Supply of Arterial Blood.—Gangrena Spontanea.—Gangrena Senilis.—Ergotism.—4. Noma.—Gangrene in Various Blood-Diseases.—Treatment.

We have already spoken frequently of gangrene and mortification. You know in general what they mean, and have already encountered a series of cases where there was local death of a part; but there are many other circumstances, with which you are not yet acquainted, which favor gangrene; all of which we shall include in this chapter.

You already know the word *gangrene* to be perfectly synonymous with mortification. Originally it was only used to express the stage where the dying part was still hot and painful; that is, not completely dead. This was called "hot mortification," while the moist "cold mortification" was called by the old authors *sphacelus*. The word *mummification* is also employed for dry gangrene. From the moment the circulation ceases, *moist gangrene* is perfectly analogous to ordinary putrefaction. Although it cannot always be certainly stated why *dry* gangrene occurs in one case and *moist* in another, we say generally that when the circulation ceases suddenly, especially if the parts have been previously inflamed or œdematous, moist gangrene occurs. Dry gangrene—mummification or shrinking of the parts—is more frequently due to gradual death, where the circulation has continued feebly in the deeper parts, and the serum has been carried off from the gradually-dying parts by the lymphatic vessels and veins. Rapid evaporation of the fluid also induces gradual dryness. It is certainly true that even in moist gangrene a superficial dryness of the skin may occasionally be obtained by removing the hard layer of the epidermis, which readily peels off from the decomposing limb; we may also greatly favor the drying by applications of substances having a strong affinity for water, such as alcohol, solutions of corrosive sublimate, sulphuric acid, etc.; but we cannot obtain so complete a mummification as sometimes occurs spontaneously. Hence, dry gangrene is not a simple putrefaction, but a rather complicated process, which gradually leads to arrest of the circulation.

The immediate cause of death of individual parts of the body is always the complete cessation of the supply of nutriment consequent on arrest of circulation in the capillaries; under some circumstances the chief arteries or veins of an extremity may be locally obstructed, and, nevertheless, the blood finds its way by neighboring branches into their lower or upper parts. Hence, obstruction of an artery can only be the immediate cause of gangrene when collateral circulation is impossible. This may be due partly to anatomical conditions, partly to great rigidity of the walls of small arteries, partly to very extensive destruction of the walls of the artery, as when the femoral is obstructed from the bend of the leg to the foot, the nutrition only ceases when the capillary circulation is rendered impossible by these circumstances. But it is not always necessary that cessation of circulation in a small capillary district, or in the parts supplied by one small artery, should cause actual decomposition; under such circumstances the disturbance of nutrition may assume a milder form, especially when this limited disturbance of circulation comes on slowly and gradually. In this case there is molecular disintegration of tissue, which shrinks and dries to a yellow cheesy mass, in short, there is a series of metamorphoses which in the cadaver appear as dry, yellow infarctions; this is essentially merely a sort of dry gangrene limited to a small spot. If this disturbance of nutrition and molecular disintegration of tissue take place on a surface, we call it *ulceration;* the whole series of so-called atonic ulcers, to which we shall hereafter return, are mostly due to such quantitative disturbances of nutrition. Hence, intimate as is the connection between the causes of dry gangrene and ulceration, still, the various forms of gangrene are well marked and peculiar, as you will see from what follows, as there is generally not only molecular disintegration of tissue, but death of whole shreds of tissue, or even of an entire limb. *A priori*, it is certainly supposable that complete closure of all the veins returning blood from a limb, should induce complete stasis in the capillaries; but in practice this is very unlikely to occur, for the veins are so very numerous, and in almost all parts of the body there are two ways for the return of blood, viz., the deep and subcutaneous veins, which communicate freely; if one way be closed, the other will be at least partly open. When dry gangrene occurs in the skin and deeper soft parts, they usually assume a grayish-black, then a coal-black hue. In cases where the parts were previously inflamed, the skin appears at first dark violet, then whitish yellow, it only becomes brownish or grayish black in case of partial drying; dead tendons and fasciæ change their color little. When, from disturbance of the circulation, a considerable portion of tissue ceases to be nourished, the border between dead and living regularly becomes more distinctly marked; around the dead skin there forms a bright-red line, the so-called *line of demarcation*. This redness is caused by distention of the capillary vessels, which is partly due to collateral circulation in them, partly to fluxion induced by the decomposing fluids, and exactly resembles the redness around the edges of a wound with loss of substance, especially of a contused wound, as we have already explained. Along with these changes in the vessels there is an active cell-infiltration in the line of demarcation, by which the tissue, whatever its nature may be, is partly softened and dissolved. All over the borders of the living tissue young cells in the form of pus appear in place of the firm tissue, and then the coherence of the parts ceases. The dead becomes detached from the living, and on the borders of the latter there is a layer of tissue changed by infiltration of plastic matter and ectasia of the vessels, granulations. To express this simply in surgical language we say: The dead tissue must be thrown off from the living by free suppuration, and this detachment of the dead tissue is followed by active granulations which cicatrize in the usual manner. This process repeats itself in all tissues, in all forms of gangrene, sometimes quicker, sometimes more slowly, in exactly the same way, even in bones, as you know from the necrosis of the ends of the bone in open fractures. But we shall not here treat of gangrene of bones, as it is so intimately connected with their other chronic diseases that we shall have to speak of it when treating of them. The time required for the detachment of the dead tissue may vary greatly. It depends: 1. On the size of the dead portion; 2. On the vascularity and consistence of the tissue; 3. On the strength and vitality of the patient.

As gangrene is usually the result of other diseases, it is not always easy to correctly group the symptoms which are to be referred to it. If the line of demarcation has formed, and the process of detachment is going on, an effect on the general health is apparent when the gangrene affects large extremities. Then there is a general marasmus, a gradual loss of strength, depression of the bodily temperature, small pulse, dry tongue, a half-soporose state in which the patient grows weaker and weaker, and finally dies, without our being able to discover in the cadaver any particular cause of death, although in other cases putrid metastatic abscesses are found in the lungs. These cases are one form of chronic septicæmia; I have no doubt that the repeated absorption of putrid matters, during the development of gangrene, by the blood and lymphatic circulation which partly continues, may be the cause of death. I propose to return to this question in the next section.

After these general remarks, we must study more carefully the different varieties of gangrene, according to their remote and proximate causes, and their practical importance:

1. Complete loss of vitality of the tissue through mechanical or chemical action, such as crushing, contusing, great heat or cold, caustic acids and alkalies, continued contact with ammoniacal urine, with carbunculous poison, poisons from certain serpents, putrid matters that act as ferments, etc., come under this head. We have already spoken of some of these varieties; we shall shortly come to others of them.

2. Complete arrest of the circulation, by circular compression or other mechanical cause, is in many cases the cause of capillary stasis and gangrene. For instance, if you surround a limb firmly with a bandage, you will have, first, venous congestion, then œdema, and finally, gangrene. Let us take a practical example: if the prepuce be too small and be forcibly drawn back over the glans so as to cause a paraphimosis, the compressed glans, or in this case more frequently the compressing ring, becomes gangrenous. The mortification of strangulated hernia depends on the same cause.

Continued pressure also, by arresting the afflux and efflux of blood, may lead to gangrene, especially in persons in whom the heart's action is weakened by long disease, or who by general septic intoxication are already disposed to gangrene.

*Decubitus*, the so-called bed-sore, is such a gangrene caused by continued pressure, but all sorts of bed-sores are not gangrenous from the first, for in some cases they are rather to be compared to a gradual maceration of the epidermis and cutis, as a result of continually lying in a bed wet with sweat, urine, and other liquids. Decubitus is particularly frequent over the sacrum, and may there attain a fearful size, all the soft parts becoming gangrenous down to the bone; it may also occur over the heel, the trochanters of the femur, head of the fibula, scapula, or spinous processes of the vertebræ, according to the position of the patient. The same thing may be caused by badly-applied dressings. This disease is the more unpleasant, as it usually comes during other exhausting affections. Although nc disease in which the patient is condemned to long, absolute quiet, is entirely exempt from the disagreeable accompaniment of a decubitus, still some peculiarly dispose to it, chief among which is typhus; in patients with septicæmia, decubitus occurs very early, often even after three to five days of quiet; it usually begins with a very circumscribed congestion of the skin over the sacrum, while, with proper care, consumptive patients keep their beds for months or years, without having bed-sores.

This disease is particularly troublesome for the patient, because, especially in chronic maladies, it may be accompanied by great pain; in acute cases of typhus and septicæmia, on the contrary, the patients sometimes do not feel it at all when they have a very large bed-sore. This form of gangrene is particularly dangerous when the exciting causes cannot be entirely removed, and it becomes progressive; the prognosis is worse the more exhausted the patient; not unfrequently bed-sore is the cause of death, as it continues to enlarge in spite of all treatment, or it may be the origin of a fatal pyæmia.

Too great *tension of the tissue*, causing great distention of the vessels, and compressing some of them, induces, on the one hand, a diminished amount of blood, while the pathological requirements of nutriment are increased; on the other, a coagulation of blood in the capillaries from the increased friction. This is the cause of gangrene occurring in inflammation, and which we have already mentioned when speaking of phlegmon, but it must not be said that every stasis of the blood in the capillaries that may occasionally occur in inflammation is to be referred to great tension of the tissues, as there are also other causes. It would lead me too far to enter on theories, especially as you have already heard them in the course on general pathology. Moreover, we shall return to this when treating of thrombosis of the veins.

3. *Complete arrest of the supply of arterial blood*, which is particularly due to diseases of the heart and arteries, must also sometimes lead to gangrene; in this class belong those cases of gangrene called *gangræna spontanea*, or oftener *gangræna senilis*, from its more frequent occurrence in old persons; this may come in various ways and forms. The causes may vary thus: The coagulation of blood may begin in the capillaries (marasmic thrombosis as a result of debility of the heart, or insufficient conduction through the smaller arteries), or as an independent thrombus of the artery, or, lastly, a thrombus from embolism; excessive, continued anæmia also, with great consecutive contraction of the arteries and debility of the heart, and, lastly, continued spasmodic contraction of the arteries, may induce gangrene. Gangræna senilis proper is a disease originally oc

curring in the toes, rarely in the fingers, as I once saw. There are two chief forms: in one of them a brown spot forms on one toe; it soon becomes black, and gradually spreads till the whole toe becomes completely dry. In favorable cases a line of demarcation forms at the phalango-metatarsal articulation, the toe falls off, and the wound cicatrizes. But the mummification may go higher and limit itself in the middle of the foot, above the malleoli, in the middle of the leg, or just below the knee. In another series of cases, the disease begins with symptoms of inflammation, œdematous swelling of the toes, very great pain, and dark, bluish-red color, which subsequently becomes black; there are stages of the disease where, by the bluish-red mottled appearance of the skin, we may see that in one place the circulation is carried on with the greatest difficulty, while elsewhere it has already ceased; this struggle between life and death the French have not inaptly compared to death by asphyxia, and termed *asphyxia locale*. In this form of moist, hot gangrene, the disease usually attacks several toes at once, and extends to the foot, till in the course of a few weeks the entire foot, perhaps also the leg, becomes gangrenous; at the same time decomposition soon begins in the œdematous subcutaneous cellular tissue, and the danger of absorption of putrid matter through the lymphatic vessels is much greater than in the process of mummification. The seat of the disease of the arteries that leads to spontaneous gangrene varies; in *acute* (marasmic) *gangræna senilis*, the primary coagulation due to feeble circulation occurs in the capillaries and thence extends backward to the arteries. The feebleness of the arterial circulation may be due to various causes: 1. To diminished energy of the heart's action; 2. To thickening of the walls of the arteries and contraction of their calibre; 3. To degeneration of the muscular coat of the smaller arteries. In some cases all of these causes unite, for, in old persons with feeble heart-action, diseases of the arteries are the most frequent; besides, affections of the heart and arteries usually have a common constitutional cause. This is not the place to discuss extensively how far rigidity and atheroma of the coats of the artery are to be referred to inflammation, or to be regarded as a peculiar disease; nor can I permit myself to discuss further the distinctions of the finer histological points, of which we shall have something to say when treating of aneurisms, but will simply mention that in old persons the coats of the arteries are oft thickened, and deposits of chalk form in them to such an extent th the whole artery is calcified and the calibre considerably dimininish by the thickening of the walls, and the inner surface becomes roug so as to dispose to the fixation of blood-clots. The original qualit of the arteries are thus lost to such an extent that they are neith elastic nor contractile, and hence, partly from the diminished calibre, partly from the lack of contractility, the onward movement of the blood, already moved less forcibly on account of the feeble action of the heart, is very much impeded, so that it is easy to understand how coagulation occurs in such cases, especially in parts distant from the heart.

While the cases just described are with some justice termed senile gangrene, and their connection with arterial diseases has been generally recognized since the time of *Dupuytren*, there is another form of spontaneous gangrene, which occurs in old persons, but is distinguished from the above, because a large portion of an extremity, as of the leg as high as the calf or the knee, becomes gangrenous at once This takes place as follows: In the chief artery, say the femoral, along the thigh or in the hollow of the knee, a firm clot forms and adheres to the wall of the vessel by rough prominences on the internal coat, due to precedent atheromatous disease, or else forms in sac-like dilatations of the artery and gradually grows by apposition of new fibrine, so as not only to fill the calibre of the artery, but to plug up the whole peripheral end of the vessel, and even a portion of the central end, by the fibrinous clot. The consequence of this stoppage of the artery by a thrombus developing on the wall, which gradually arrests the *collateral* circulation also, is usually gangrene of the whole foot and part of the leg, which is dry or moist according to the rapidity with which the clot has developed; it is occasionally possible to trace the growth of the thrombus by the spread of the gangrene. Not long since I observed an old man, who was taken into the hospital for spontaneous gangrene of the foot. He was so thin and the arteries were so rigid that the pulsations of the femoral could be distinctly followed into the hollow of the knee. Subsequently the gangrene progressed, and at the same time the pulsation in the lower part of the artery ceased. About a fortnight later, shortly before death, when the gangrene had advanced to the knee-joint, the pulsation had ceased at Poupart's ligament. The autopsy confirmed the diagnosis of complete arterial thrombosis. The gangrenous leg was so completely mummified that I cut it from the body, and, to preserve it from further destruction and worms, varnished it. It is still in the surgical museum at Zürich.

Another case of arterial thrombosis is where the primary stoppage of the artery is caused by an embolus. A clot of fibrine, in endocarditis or detached from an aneurismal sac, may become wedged in an artery of one of the extremities; this induces further deposit of fibrine. Of late, there is a tendency to refer most cases of softening and desiccation, as of the brain, spleen, etc., to such emboli. In our clinic we saw a very interesting typical case of this variety. Six weeks after confinement, a young woman had great swelling of the left leg, which was soon followed by a dark-blue color of the skin, and complete putrefaction of that part of the body; there was general septic poisoning when the patient entered the hospital. As there was no excessive anæmia, and no disease of the arteries could be discovered, I made the diagnosis of endocarditis with fibrinous vegetations on the mitral valve, and detachment of one of these vegetations, with its lodgment at the bifurcation of the left popliteal artery. I held to this diagnosis, although no abnormal murmur could be discovered, for it is well known that some cases of endocarditis run their course almost without symptoms; the rapid putrefaction of the leg must have had a sudden cause. As no line of demarcation formed, and the general condition daily became worse, we could have no hopes of saving life by amputating; death took place about twelve days after the first symptoms of gangrene; the autopsy fully confirmed the diagnosis. It seems remarkable that no collateral circulation should develop in such cases, as it does after ligation of the femoral artery. I can only explain this on the supposition that in endocarditis the heart's action is weakened, and consequently the pressure of the blood is insufficient to dilate the smaller collateral arteries.

Very rare are the cases where from excessive anæmia the arteries are so much contracted that but little blood circulates through the smaller ones, and the nervous excitation of the heart is so slight that its contractions are incomplete. Cases of spontaneous gangrene from this cause are more frequent in slender chlorotic females than in men; the patients, who are generally young, often suffer from rigidity of the hands and feet, fainting-fits, and fatigue. This disease appears to be more frequent in France than in Germany or England. There is an excellent work on the subject by *Raynaud*, entitled "De l'asphyxie locale et de la gangrene symétrique des extrémités," 1862. As implied by the title, the gangrene is usually symmetrical in the two limbs. I have only seen one such case; a young, very anæmic man, without any apparent cause, had first gangrene of the tip of the nose, then of both feet. After suffering for months, he died; as on the patient, so on the cadaver, I could find nothing morbid beyond the excessive, inexplicable anæmia.

The form of gangrene seen from eating spurred rye is referred to permanent spasmodic contraction of the smaller arteries; experience shows that this substance induces contraction of the organic muscular fibres, especially of those of the uterus, and it is supposed of the uterine arteries also.

*Spurred rye, secale cornutum*, is a diseased grain growing in the ear of rye (secale cereale), in which is developed a peculiar material.

ergotin. If bread be made from such grain, persons eating it are affected with peculiar symptoms, which are comprised under the name *ergotismus* or *raphania*. As the above disease of the grain is usually limited to certain regions, it may be readily understood that the disease should occur epidemically in men and beasts. It has been known for a long time, but the first accurate descriptions are of an epidemic in France in 1630. The disease seems to have occurred rarely in Germany, England, or Italy. Of late it hardly ever occurs, probably because the diseased grain is better known and is no longer used for food, and because less of the grain is grown since potatoes have come into common use. From former descriptions, various forms and courses of the disease may be distinguished, of which sometimes one and sometimes another prevailed in the different epidemics; possibly the poison is not always the same, or is at least of variable intensity. In the acute cases, the patients were soon attacked with severe general cramps, and death resulted in from four to eight days; cramps only occur occasionally; at the same time, and previously in the prodromal stage, there are great itching and crawling in the skin, but particularly in the hands; there is also a feeling of deadness, of anæsthesia in the ends of the fingers, rarely moist gangrene of the skin, then of whole extremities. In more chronic cases, the result is usually favorable, although several fingers or toes may be lost.

4. We have still to speak of several forms of gangrene whose causes are not exactly known, in which probably several influences unite. Among these is so-called water-canker, *noma*, a spontaneous gangrene of the cheeks, especially common in children, which is most frequent in cities along the Baltic, and more rare inland. Very puny children, living in cold, damp dwellings, are particularly prone to this disease, in which, without any known cause, a gangrenous nodule forms in the middle of the cheek or lip and spreads rapidly till the child finally dies of exhaustion. It is doubtful whether this is due to anæmia with feebleness of the heart, to miasmatic influence, or to some peculiar disease of the blood. In occasional remarks about septicæmia, we have already stated that certain morbid states of the blood predispose to gangrene. Under this cause we must class the cases occurring after typhus, intermittent and exanthematous fevers, in diabetes mellitus, morbus Brightii, etc. After and during these diseases, gangrene of the tip of the nose, of the ear, cheeks, hands, and feet, occurs; and in rare cases an exanthema of the skin may pass into gangrene. In such cases we may consider that the miasma which has induced the constitutional disease also influences the occurrence of the gangrene; and, on the other side, there seems reason for the idea that these cases are mostly the result of feeble action of the heart.

induced by the long illness, which proves insufficient to carry the blood to the remote parts of the body with sufficient energy; according to this view, this gangrene would be due to marasmic capillary thrombosis. Doubtless various circumstances act more or less prominently in individual cases, so that no definite etiology can be given for these rare forms of gangrene from internal causes. I may also mention that stomatitis, from excessive use of mercury, also has a great tendency to gangrene. We shall hereafter speak of a peculiar form of gangrene of wounds, the so-called *hospital gangrene*.

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There are certain important prophylactic rules for the prevention of gangrene, especially of decubitus and other forms due to pressure; even gangrene from inflammation may sometimes be prevented, by relieving the great tension of the tissue and the venous congestion by an incision made at the proper time. Be constantly on your guard against bed-sores in all diseases at all disposed to decubitus; turn your attention to this point early: a well-stuffed horse-hair mattress is the best sick-bed; the sheets placed over it should always be kept smooth, so that the patient shall not lie on wrinkles. As soon as any redness appears over the sacrum, you should be doubly careful about the passages of urine and fæces, so that the bed may not be wet. Let a lemon be cut and the reddened spot rubbed daily with the fresh juice from the cut surface. If there be excoriation over the sacrum, place the patient on a ring cushion, or, if possible, on a caoutchouc, air, or water cushion. The excoriation may be painted with nitrate of silver, or covered with leather spread with lead-plaster. If the decubitus be gangrenous from the first, and this begins to extend, we should resort to the ordinary treatment of gangrene, of which we shall speak presently.

The *local treatment* of gangrene has two chief objects: 1. To promote detachment of the gangrenous parts by exciting active suppuration, which is accompanied by arrest of the gangrene; 2. To prevent the gangrenous parts decomposing, and thus acting injuriously on the patient, and infecting the chamber too much.

For the first indication, moist warmth in the form of cataplasms was formerly employed. But I cannot find that they are peculiarly efficacious in these cases. If the gangrene be moist and the gangrenous parts are much inclined to decompose, this would only be favored by the application of cataplasms; for the detachment of a dry eschar, which does not smell badly, and when the line of demarcation is already formed, it is hardly worth while to hasten the process a little by warmth. Hence I prefer covering the gangrenous parts and the borders of the healthy tissue with compresses or charpie, soaked in chlorine-water, and thus in moist gangrene I also diminish the bad smell of the decomposing substances. For the same purpose, we may use creosote-water or carbolic acid, or dilute purified pyroligneous acid, very strong alcohol, spirits of camphor, or oil of turpentine. Charcoal-powder absorbs the gases from the decomposing substances, but, as it soils the parts very much, it is perhaps too little used. Other powerful antiseptics are acetate of alumina (alum ʒ v, plumbum aceticum, ℥ j, aqua, ℔ 1), and coal-tar with plaster; both remedies are very serviceable, but, like all similar ones, must be freshly applied several times daily to remove entirely the smell of the decomposing parts. Of late, permanganate of potash (gr. x to ℥ i water) has been greatly praised as a local antiseptic and disinfectant; I have made several trials of it, but have found it far inferior to the remedies previously mentioned. Concentrated solutions of carbolic acid in olive-oil (say ʒ ij to ℔ 1) cause symptoms of poisoning (olive-green urine), hence they should be used carefully. As soon as the gangrenous mass has become somewhat detached, the shreds should be removed with the scissors, without cutting into the healthy parts; this is particularly important in gangrene of the subcutaneous cellular tissue, which is often extensive, as after infiltration of urine; at the same time the local antiseptics should be continued till healthy granulations arise. Led by the anatomical conditions in spontaneous gangrene, it has been advised to break up the coagulation of blood, by stroking and rubbing the limb; from the pain and swelling of the parts, this is rarely practicable; in cases where I have had it done, it has had no effect on the progress of the gangrene.

If the gangrene affect a limb, as in the various forms of spontaneous and senile gangrene, I strongly urge you not to do any operation till the line of demarcation is distinct. If there be merely gangrene of single toes, leave their detachment to Nature; if the whole foot or leg be affected, do the amputation so that it may be merely an aid to the normal process of detachment, i. e., on the borders of the healthy parts you try to dissect up only enough skin to cover the stump, and saw the bone as near as practicable to the line of demarcation. Thus you will occasionally succeed in avoiding a new outbreak of the gangrene, and in saving your patient's life. If the patient dies before a distinct line of demarcation has formed (as is frequently the case), you need not reproach yourself for having neglected amputation, for you may rest assured that the patient would have died even sooner if amputation had been performed. The prognosis in gangrene from internal causes (as the older surgeons termed it) is generally bad.

The *internal* treatment should be strengthening, in some cases even stimulant. Nourishing food, quinine, acids, and occasionally a few doses of camphor, are proper. The severe pain in senile gangrene often calls for large doses of opium, or subcutaneous injection of morphine. For gangrene in stomatitis, after poisoning by mercury, we have no decided antidote; the use of the mercurial should be at once stopped; if mercurial salve has been employed, the patient should be bathed, placed in a fresh, airy chamber, provided with clean body and bedclothes, and have a gargle with chlorate of potash or chlorine water. Nor have we any antidote for ergotin, which causes raphania; emetics, quinine, and carbonate of ammonia are chiefly recommended. We could only put off the continued absorption of putrid matter into the blood, by amputation; but we have already mentioned that this is a very precarious remedy in spontaneous gangrene.

At the opening of this chapter I said, "The immediate cause of death of individual parts of the body is always the complete cessation of the supply of nutriment, mostly due to arrest of circulation in the capillaries." This admits the possibility of gangrene occurring in tissues where capillary circulation still continues. Formerly this seemed to me impossible; I could not imagine a dead gangrenous tissue with capillary circulation. But observations at the bedside, together with the impressions derived from *Samuel's* investigations of inflammation, have made it seem to me probable that the inflammatory disturbance of nutrition of which we have spoken occasionally starts up and extends so rapidly that it leads directly to arrest of the vital change of tissue, even before the occurrence of stasis and coagulation in the capillaries; the blood then circulates in tissues which fulfill no normal function of interchange of tissue, but in which the juices are being decomposed in an abnormal way, which may even be the same as putrefaction. Paronychiæ and more rarely phlegmons sometimes pass so rapidly to gangrene, that from analogy with other processes it seems very improbable that they should be due to arterial thrombosis; when the tissue has become gangrenous, the capillary circulation soon ceases, but not from disturbance of the circulation in the arteries and veins, as in gangrene from incarceration, but from arrest of function of their walls from the inflammatory process, which I regard as a higher grade of the inflammatory alteration (*Cohnheim*) that takes place rapidly in such cases. It seems that such a rapid change from inflammatory alteration to destruction of tissue is particularly apt to follow septic poisoning; possibly snake-poison acts in the same way. More of this hereafter. Here we must again mention the fibrinous infiltrations of the cellular tissue (diphtheritic phlegmons). Some clinical observations seem to show that fluid blood can continue to flow for a time through the vessels of tissues whose juices have nearly hardened, that in some of these cases the thrombosis is only the result of the infiltration of tissue, and that the tissue sometimes dies before the circulation is fully arrested. At present we cannot fully explain this symptom ; I only wished to induce you when opportunity offered to attend to these practically important processes. The view is not new, for the old surgeons regarded gangrene as the highest point of inflammation.

[Quite recently Dr. *F. von Winiwarter* has described a case of spontaneous gangrene which has hitherto probably been classed with atheromatous embolism. The patient was fifty-seven years old, and for years he had suffered from pains regarded as rheumatic, and then gradually gangrene of the toes and of the foot came on, and made amputation of the thigh necessary. Examination of the vessels showed proliferation of the intima of the arteries and veins, commencing in the largé trunks and not leading to any retrograde metamorphosis, terminating in a cellular fibrous mass obliterating the vessel. The media and adventitia hardly participated in this process. The affection would be a primary obliteration of the large vessels by a peculiar form of endarteritis and endophlebitis, which it is practically important to recognize, as in certain cases it might induce us to amputate in healthy tissue before the line of demarkation is formed, which is not the custom in cases of ordinary senile gangrene. The case above mentioned recovered ; so this mode of treatment gives some hopes.

It has been previously stated that in some persons cold causes spasmodic contraction of the arteries which induces temporary complete anæmia, usually only in certain fingers. Such symptoms have also been observed in patients in whom spontaneous gangrene developed. If, from any cause, the spasm of the arteries does not subside the anæmic limb must become gangrenous. These patients, who are mostly youthful, also have other symptoms of disturbed circulation, such as faintness from cerebral anæmia, continued chilliness, great depression, etc.

It is not always the peripheral portions of the extremities that are attacked by gangrene, but this may occur in the skin, perhaps at symmetrical parts of the arm (from disease of the nerves of the vessels) ; demarkation occurs, eschars fall, and the loss of substance heals by granulation. This may occur occasionally, while in the interval temporary disturbances of circulation are common.

When a patient has to lie steadily on the back, the skin over the sacrum and buttocks should be examined daily and washed with equal parts of vinegar and water, or, if it has become red, it may be rubbed with a piece of lemon.

When gangrene has occurred, *Lister's* dressing may be applied. It has been advised in the commencement of the disease to break up the blood-clots by rubbing; but this causes too much pain, and may start off emboli to other parts.

If gangrene develops and extends very slowly, while the general condition of the patient is good, there is probably disease of the arteries; there is no exact limit to the gangrene, still, in these cases, amputation is indicated while the general condition would render it endurable; but it should be done through healthy tissue, at some distance from the gangrene.

The form of gangrene called noma requires particularly energetic treatment to arrest the progress of the disease: we should remove the dead portion, and induce active reaction in the healthy tissue. First the gangrenous part, usually on the cheek, is to be divided by a deep incision and the infiltrated tissue scraped out with a sharp scoop, especially about the gums, on whose border the gangrene often creeps into the alveolar process. After removing all that can be taken away with the scoop and arresting the hæmorrhage, apply fuming nitric acid to the raw surface, so as to cause a firm, deep slough; then fill the wound with charpie soaked in acetate of alumina, and over this apply moist warmth, and change this dressing twice daily. If the infiltration of the cutis progresses, touch the parts with tincture of iodine. The gangrene may thus be arrested in some cases, and life preserved. The loss of substance must be replaced by plastic operations.]

# CHAPTER XIII.

## *ACCIDENTAL TRAUMATIC AND INFLAMMATORY DISEASES, AND POISONED WOUNDS.*

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### LECTURE XXIV.

I. Local Diseases which may accompany Wounds and other Points of Inflammation: 1. Progressive Purulent and Purulent Putrid Diffuse Inflammation of Cellular Tissue.—2. Hospital Gangrene, Ulcerative Mucous-salivary Diphtheria, Ulcerative Urinary Diphtheria.—3. Traumatic Erysipelas.—4. Lymphangitis.

GENTLEMEN: When speaking of traumatic inflammation, I told you that it did not extend beyond the bounds of the injury, and that this was only apparently the case when we could not accurately examine the injured part. I still maintain the truth of this. But we have already added that, from various accidents, either immediately after the injury, as in contused wounds, there may be very severe progressive inflammation, with putrefaction, or that, later, secondary inflammations may develop around the already granulating wound from causes which we mentioned at the time (Lecture XIII.). I must now tell you that still another series of peculiar, partly inflammatory, partly gangrenous processes occur in the wound, which cause severe, usually feverish, constitutional diseases. Some of the latter may also occur without any thing peculiar being observable in the wound. Lastly, substances may enter a wound already existing, or at the time of its occurrence (as from the bite of a poisonous or diseased animal), which may induce both severe local inflammation and general blood-poisoning. In this chapter I shall speak of all these things; I will try to give you a general view of them. We shall speak first of the local symptoms which accidentally accompany a wound, or an inflammation due to other causes.

## I. LOCAL DISEASES WHICH MAY ACCOMPANY WOUNDS AND OTHER POINTS OF INFLAMMATION.

1. For the sake of completeness, we here mention again progressive suppurative and sanio-purulent diffuse inflammation of the cellular tissue. Putrid matters which form on fresh wounds from gangrene of the surfaces of the wound, and may diffuse rapidly in the meshes of the cellular tissue, occasionally cause, on the second, third, or fourth day, those forms of inflammation of the cellular tissue that are characterized by rapid decomposition of the inflammatory product and by rapid extension. If the patient survives the demarkation of such a phlegmon, the process always ends with necrosis of the infiltrated cellular tissue and panniculus adiposus. The same thing occurs in fibrinous (diphtheritic) phlegmon. Both processes are usually accompanied by severe constitutional symptoms. If suppuration has already begun, as long as the wound is open, phlegmonous inflammation may spread around the wound from mechanical irritation, foreign bodies, great congestion, retention and decomposition of pus in the recesses of the wound, or infection of the wound with phlogogenous substances of various sorts (Lecture XXI.).

2. *Hospital Gangrene, Gangræna Nosocomialis; Pourriture des Hôpitaux.*—I will first describe the disease, then add a few remarks about the etiology. At a certain time we notice, especially in hospitals, that a number of wounds, as well those from recent operations as those that were granulating and cicatrizing, without known cause, become diseased in a peculiar manner. In some cases the granulating surface changes partially or entirely to a yellow smeary pulp, which may be washed off from the surface, but more deeply it is firmly adherent. This metamorphosis extends not only to the granulating surface, but to the surrounding skin which was previously healthy, which becomes rosy-red; this also assumes a smeary yellowish-gray color, and in from three to six days the surface of the original wound almost doubles. The increase in depth is less in the so-called *pulpous* form of hospital gangrene. In other cases a fresh wound, or a granulating surface, rapidly assumes a crater shape, excretes a sero-putrid fluid, after the removal of which the tissues lie exposed. The surrounding skin is slightly reddened. The progress of this molecular disintegration to thin ichor is usually in sharply-cut circles, so that the wound may acquire a horseshoe or trefoil shape. This *ulcerous* form of hospital gangrene progresses more rapidly than the pulpous, and extends with especial rapidity in *depth*. Although both of the above forms occasionally occur separately, they are also seen in combination. I have seen the pulpous form oftener than the ulcerous, but acknowledge that my individual experience of diphtheria of wounds is based on a small number of ob-

servations. Hospital gangrene does not attack chiefly large wounds, but rather insignificant injuries, such as leech-bites, cup-cuts, even the portions of skin denuded by a blister, while it never occurs on an uninjured part of the skin. The resemblance to diphtheritic inflammation of the mucous membranes is mentioned by some authors. But after seeing a wound infected from a diphtheritic mucous membrane, I am convinced that diphtheria and hospital gangrene are two different processes. A wound attacked by diphtheria is covered with thick fibrinous rinds; the entire wound becomes infiltrated and the surrounding parts intensely erysipelatous; then a large part of the infiltrated tissue becomes necrosed and breaks down or falls off in shreds. But we do not see the daily progress of pulpous degeneration, forming round figures on the margins of the wound, which are puffy, very sensitive, and inclined to bleed, as is so common in hospital gangrene. It is well known that after diphtheria of mucous membranes paralysis is not unfrequent; but this has not been observed after hospital gangrene. In the latter disease there are at the same time constitutional symptoms: at first the fever is not generally severe, but there is more or less gastric affection; the tongue is coated, there is inclination to vomit, and general depression. The disease may prove dangerous to old or debilitated persons, especially if it eats away small arteries and causes arterial hæmorrhage. The large arteries often resist hospital gangrene wonderfully. I once saw a man, for whom an inguinal abscess had been opened, attacked by the pulpous form of the disease; the skin of the groin to about the size of the hand was destroyed; the disease had advanced so deep that about an inch and a half of the femoral artery lay exposed in the wound, and could be distinctly seen pulsating. I detailed a nurse to stay with the patient constantly, and to make instant compression if bleeding should occur, as it might at any moment. The pulp was thrown off, the wound granulated rapidly, and after a long time complete recovery took place without hæmorrhage. The erysipelatous redness accompanying diphtheritic phlegmon and hospital gangrene is occasionally as sharply bounded and desquamates as much as in erysipelas about wounds otherwise healthy; but it has not the same tendency to spread. The constitutional septic poisoning is worse in diphtheria than in hospital gangrene.

Views as to the causes of hospital gangrene vary; this is chiefly because many living surgeons have had the good or bad fortune never to have seen the disease; thus in Zürich it has never been seen. In his maxims on military surgery *Stromeyer* states, as a young physician in the Berlin Charité, he had only seen one case of hospital gangrene. Surgeons who have not seen this disease, or have only seen sporadic cases, think it is due to gross neglect, dirty dressings, etc., and regard it as little more than an ulcer of the leg that has superficially become gangrenous from dirt and neglect. Other surgeons suppose that hospital gangrene is, as the name would indicate, a disease peculiar to some hospitals, and that its occurrence is only promoted by neglect of the dressings. Lastly, a third view is that this form of gangrene is due to epidemic influences, and that its name is in so far incorrect as it occurs outside and inside of hospitals at the same time. In the hospitals it probably spreads by inoculation, for I do not doubt that matter can be carried from gangrenous to healthy wounds by forceps, charpie, sponges, etc., and there excite the disease. *Von Pitha* and *Fock* have expressed the belief that it is an epidemic-miasmatic disease. In the surgical clinic at Berlin with *Fock* I observed an epidemic, while the disease was seen not only in other hospitals in Berlin, but in the city, in patients who could not be proved to have had any thing to do with a hospital. The disease appeared very suddenly, and entirely disappeared in a few months, although the treatment of the wounds had not been at all changed, nor could any changes be made in the hospital itself. This seems to show that the causes do not lie in the hospital itself. Epidemic hospital gangrene might occur from certain small organisms, which are rarely developed, which, like a ferment, induce decomposition in the wound and granulating tissue; hence I should preferably compare this disease of wounds with blue suppuration, which causes no injury to the wounds, but, according to *Lücke*, like blue milk, is caused by small organisms and can infect other wounds. The requirements for the growth of these small bodies are probably particularly favored by certain atmospheric influences; hence the disease spreads epidemically. There is no doubt that in the pulp of every hospital gangrene micrococci and streptococci are just as frequent as in the secretion of simple diphtheritic wounds. But it has not been proved that they were in the tissue before it was destroyed, that they grew in it, or broke it down into pulp; nor has it been shown that this is a peculiar variety of micrococcus. But it is certain that the transfer of hospital gangrene pulp or putrid matter to healthy wounds usually (always, according to *Fischer*) induces hospital gangrene, and this is very important in practice. From my recent experience in the Vienna General Hospital, I am more and more convinced that this disease results from specific causes, entirely independent of pyæmia, septicæmia, erysipelas, and lymphangitis, although it may be followed by either of these diseases.

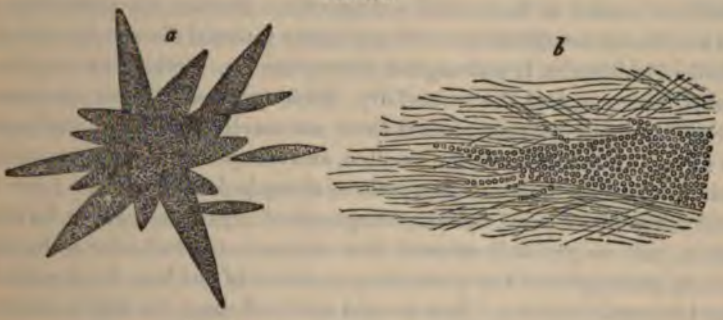
The first point in the treatment is strict isolation of the patients, who should have special nurses, dressings, and instruments. If this does not entirely prevent the spread of the disease, as the contagion may possibly be carried by the air from a diseased to a healthy wound, still experience shows that it interferes with the spread. In some epidemics in military hospitals it was necessary entirely to vacate certain localities. Locally we should apply strong chlorine-water, or spirits of camphor or turpentine, to these wounds. Sometimes painting the part every two hours with tincture of iodine acts well, or solution of acetate of alumina applied on a compress till the wound becomes clean; but the solution should not be too concentrated, and its application should be stopped when the process ceases to spread. If this also prove ineffectual, it has been recommended to burn the wound down to the healthy tissue, so that the slough shall remain attached six or eight days, as in a healthy wound. I find it just as effectual to cauterize the wound with fuming nitric acid or carbolic acid, but these cauterizations also should extend to the healthy borders of the wound, and be repeated till the slough remains adherent. The general treatment should be strengthening, or even stimulant. The fever occurring in hospital gangrene is due to reabsorption of putrid matter, and does not differ from other forms of putrid fever.

The pulpous phagedenic gangrene above described is especially apt to occur in wounds of the mouth or urinary bladder, even without any external source of infection. I mention this here because these diseases are doubtless allied to phagedenic diphtheria, although, from their limitation to certain parts of the body, they more properly belong to special surgery and the clinic. After extirpation of large portions of the tongue and resection of the lower jaw, I have sometimes seen a rapid pulpy breaking down of the wound following hard and extensive infiltration of the cellular tissue; here there is a combination of diphtheritic phlegmon with phagedenic ulceration. Most of these cases ended fatally from septicæmia; others recovered after the whole cellular tissue had become necrosed and been thrown off by free suppuration. Although mucus and saliva coming in contact with these wounds may have no phlogogenous or septic qualities of their own, putrid ferments may be mixed with them, such as are occasionally found in the coating on the gums and between the teeth of patients who do not clean their mouths habitually, or neglect this on account of painful ulcers in the mouth. So this ferment will be carried to wounds in the mouth by the mucus and saliva, thus justifying the name *mucous salivary diphtheria*. This disease only threatens the patient during the first five days after operation; only recent wounds in the mouth are infected by the ferment in question; if good granulations have once developed, this diphtheria does not occur, unless there be infection from without, or the wound be mechanically injured and the granulations partly destroyed. In this disease the constitutional symptoms may be very severe, and the patients are particularly subject to sudden collapse, which is the more dangerous as, from the impairment of nutrition which has often gone before, the patients are usually much debilitated.

After operations for stone, urethrotomy, vesico-vaginal fistula, or ectopia vesicæ, pulpous breaking down of the edges of the wound, with fibrinous coating of the walls of the bladder or of the vagina, is not rare, especially when the urine is alkaline. As this disease is associated with decomposition of the urine, it is called *urinary diphtheria.* This form of diphtheria is the mildest of those above mentioned, from having little tendency to spread, and running its course without constitutional symptoms, if the wound is kept clean. Rarely the mucous membranes break down, but more frequently the process becomes a purulent retroperitonitis, which becomes a peritonitis and causes death. Diphtheritic inflammation of the vagina also may spread as superficial suppuration to the inner surface of the uterus, and thence through the oviducts to the peritoneum; this suppurative peritonitis also is usually fatal. Under such circumstances I have never seen fibrinous inflammations. In the latter cases, which unfortunately are not rare after confinement, but do not often occur after operations for vesico-vaginal fistula, severe constitutional symptoms are early manifested.

In the pulp from mucous-salivary and from urinary diphtheria micrococci and streptococci are constantly found; they are just as regularly found in the coating of the gums and tongue, and in urine which has become alkaline, but seem to develop with particular rapidity in this pulp. The contagious principle of this pulp has not yet been separated from the micrococcus; so we may suppose the latter has in or on it the contagious material. There is no proof that micrococci from any source can *excite* this process; but many observations tend to show that these vegetations take up contagious substances very readily, and so become vehicles of contagions and ferments. If we inoculate the cornea of a rabbit with a fluid containing micrococci, the interesting experiments of *Nassiloff*, *Eberth*, *Leber*, *Stromeyer*, *Dolschenkow*, *Orth*, *Frisch*, and others show that the coccus grows to a certain point, and in some cases (when unaccompanied by any peculiarly injurious substances) causes irritation, chiefly mechanically, by separating the corneal lamellæ, so that the coccus colony gradually becomes enveloped in pus, and then is thrown off with the pus; but in other cases (if the inoculated matter has very deleterious properties) the whole cornea may become gangrenous in twenty-four hours, and the growth of the coccus hardly be as great as in the first instance. Lastly, cases occur where the coccus growth induces no reaction in the cornea, but disappears without leaving a trace; this is even the rule in inoculating the cornea of the dog.

FIG. 66 A.



*a*, Fungus from the cornea of a rabbit; coccus proliferation between the lamellæ of the cornea, induced by inoculation; slightly magnified. *b*, One point of *a*, magnified 600. After *Frisch*.

From this it follows that the intensity and nature of inflammations induced by such contagions do not depend on the coccus proliferation itself, but on the injurious qualities of the matter conveyed with the coccus.

I thought you should be told these things, so that you would have some knowledge about processes which are now so much discussed. I recommend for your special study the excellent monograph on hospital gangrene by *C. Heine*.

3. *Erysipelas traumaticum.* Erysipelas, as previously mentioned (page 304), is classed among the acute exanthemata, and is characterized by a diffuse swelling, rosy redness of the skin, and pain, as well as by the accompanying fever, which is usually severe. Erysipelas has a peculiar relation to the other exanthemata; on the one hand, because it often accompanies wounds, although it may apparently come spontaneously; on the other hand, because it does not generally spread by such an intense contagion as measles, scarlatina, etc.; lastly, also because, when one has had this disease, he is not only not safe from another attack of it, but in some cases is even peculiarly predisposed to it. As I dare hardly assume that you have already studied skin-diseases carefully, we will here briefly review the symptoms of this disease.

Its commencement may vary by the fever preceding the exanthema, or by their simultaneous appearance. Suppose you have a patient with a suppurating wound of the head, and after he has been previously well, and the wound was healing nicely, you find him with high fever, which may have been preceded by a chill; you examine the patient, and can find nothing but some gastric derangement, as evinced by a coated tongue, bad taste in the mouth, nausea, and loss of appetite. This state is present at the onset of so many acute diseases that you cannot at once make a diagnosis. Besides the possibility of an accidental complication with any acute internal disease, you would think of phlegmon, lymphangitis, and erysipelas. Perhaps twenty-four hours later you find the wound dry, discharging a little serous secretion; for some distance around there are swelling, redness, and pain, or the granulations are large, swollen, and croupous; the redness of the skin is of a rosy hue and everywhere *sharply bounded;* the fever is still tolerably intense; now the diagnosis of erysipelas cannot be mistaken, and we are well content that we have to deal with a disease which, although not free from danger, is one of the less dangerous of the traumatic diseases. In a second series of cases the erysipelas appears with the fever. We may for a brief period doubt whether the case be one of lymphangitis, inflammation of the subcutaneous cellular tissue, or of erysipelas, but the course of the disease will soon show this; the extent that the erysipelatous inflammation of the skin has the first day rarely remains the same, but it usually spreads farther and farther, in such a way that the rounded, tongue-shaped, projecting borders of the inflamed skin are always sharply bounded, and we can accurately follow its removal from one side to the other; in many cases the redness advances like fluid in bibulous paper. Thus the process may extend from the head to the neck, thence to the shoulders, or the anterior part of the trunk, or even pass down the arm, and finally may even reach the lower extremities. *Pfleger* has observed that the mode of extension of wandering erysipelas is almost always the same, and is probably due to the flow of certain fluids (lymph), which again depends on the arrangement of the filaments of the cutis. As long as the erysipelas spreads in this way, the fever usually remains at the same height, and thus old or debilitated persons are readily exhausted. Most cases last from two to ten days; it is rare for one to continue over a fortnight; the most protracted case I have seen was one lasting thirty-two days and recovering. In this *erysipelas ambulans* or *serpens* you will notice that the same grade of inflammation of the skin only continues a certain length of time in one place, so that when the erysipelas advances, the whole surface is not inflamed at once, but only a part at a time is at the acme of the local inflammation.

After the inflammation has remained at the same point about three days, the redness grows less, the skin desquamates, partly as a bran-like powder, or in scales and tags of epidermis. In some cases, even at the commencement of the erysipelas, the epidermis rises in vesicles, which are filled with serum (*erysipelas bullosum*). But this erysipelas is not a peculiar form of the disease; it only indicates rapid exudation. We not unfrequently see vesicles appear on the face in erysipelas, while on the rest of the body the disease has the usual form. If erysipelas attacks the scalp, the hair often falls, but grows again quickly. According to my experience, the disease is most frequent on the lower limbs, then on the face, upper extremities, breast and back, head, neck, and belly. This scale of frequency probably depends on the proportionate numbers of injuries in the different parts of the body.

Erysipelas, like other exanthemata, may be accompanied by various internal diseases, as pleurisy, and erysipelas capitis by meningitis; but, on the whole, these complications are rare, and when they occur are usually a result of the disease advancing to the deeper parts.

The course of erysipelas is usually favorable. Of one hundred and thirty-seven cases of the uncomplicated disease, which I observed in Zürich, ten died; children, old persons, and patients debilitated by previous disease are most endangered, and, according to my experience, they usually die of exhaustion from the continued fever; on autopsy, we find no remarkable change of any organ that can be regarded as the cause of death. Cloudy swelling and partial granular degeneration of the liver, kidneys, and epithelium, and softness of the spleen, are found in cases of fatal erysipelas, as after all intense blood-diseases. The nature of erysipelas is not fully understood, as its cause and the mode of its progress are not quite clear. Dilatation of the capillaries of the cutis, serous exudation in the tissue itself, and an active development of the cells of the rete Malpighii are all we can find *anatomically*. The disease rarely extends to the subcutaneous cellular tissue; it is true, this swells enormously in some places, as in the eyelids and scrotum, being greatly saturated with serum; but in most cases this œdema recedes without any sequelæ. In rare cases this œdema attains such a grade that, as a result of the great distention of tissue, the circulation of blood is arrested, and the parts (as the eyelids) may become wholly or partly gangrenous. Should all the skin of an upper or lower eyelid be lost in this way, it would cause great deformity; but usually only small portions mortify, and, in the upper lid particularly, the skin is so plenty in most persons that the defect is subsequently but little noticed. In other cases, after the subsidence of the erysipelatous inflammation, there remains a swelling of the subcutaneous tissue, in which we may distinctly feel fluctuation, and by incision may evacuate pus. Microscopic examination of skin affected with erysipelas shows only more or less infiltration of the cutis and subcutaneous tissue.

The causes of erysipelas evidently vary; that occurring without a wound, spontaneous erysipelas capitis, is said to come most frequently after catching cold. Some old persons are said to have this disease every year, in spring or autumn; psychical influences are also blamed for it, especially terror, particularly in women during their menses. I cannot vouch for the latter, but think it may belong to medical traditions. Disturbances of digestion are also regarded as causes. I am very skeptical of all the views which are not based on accurate observation, but rest on tradition; indeed, I consider it doubtful whether erysipelas ever occurs without having started from a wound or some point of inflammation already existing.

From what I have seen of erysipelas traumaticum, my idea concerning it is as follows: I consider the local affection as an inflammation of the cutis, in which the inflammatory irritation gradually spreads through the lymphatic net-works; the way in which the inflammatory redness spreads and is sharply bounded shows positively that the process is limited to the vascular districts; by close observation we may see that very often, close to the border of the redness, there forms a red, round spot, at first circumscribed, which soon unites with the previously-reddened portions of skin; these newly-forming red spots evidently represent vascular districts; we see something similar when we inject the skin through an artery; then, too, the color from the injection first appears in spots, and only unites when heavy pressure is made on the syringe; now, as the venous and lymphatic districts in the skin are to some extent analogous to the arterial, the irritating poison causing the dilatation of the blood-vessels might circulate in one of these tracts. The arterial and venous tracts in the cutis have few connecting branches parallel to the surface, while the lymphatic vessels have very many, and but few branches going down into the subcutaneous tissue; thus the exciting poison may readily spread superficially in the cutis, like liquid in bibulous paper, but it also enters the subcutaneous lymphatics, and often causes inflammation there, as well as in the neighboring lymphatic glands, striated redness of the skin, and swelling of the adjacent lymphatic glands. When I here speak of a septic or other similar poison as a cause of erysipelas, I refer only to traumatic erysipelas, for I think I have satisfied myself by observation that this is always of toxic origin. Concerning the nature of this poison, I may say: 1. It is chiefly blood mixed with decomposing secretion from the wound that induces erysipelas, which then appears the second or third day after the injury or operation. 2. There is probably a dry, dust-like substance, which, coming on the wounds, whether fresh or granulating, causes erysipelas; this substance clings especially to sponges and dressings. I have often observed that patients operated on after each other, under the same circumstances, in the same operating-room, all had erysipelas on the fresh wounds a few hours after the operation, without retention of secretion from the wound, although they lay in perfectly separate wards of the hospital. Erysipelas thus becomes domesticated in the hospital; the infecting substance may be transported on the clothes of the surgeons making the dressings; it may adhere to instruments, beds, or even to the walls. The more accurately I examined the cases of erysipelas in the Zürich hospital, and in my clinic in Vienna, the more evident was its occurrence in groups—an occurrence entirely independent of all other morbid influences outside of the hospital. From statistics during two years, supported by contributions from the physicians of the Canton Zürich, I have found that during that time erysipelas had not occurred epidemically in the country or city, but that, like other acute diseases, it was particularly frequent in autumn and spring; hence erysipelas epidemics in hospital must depend on circumstances that are to be sought in the hospital itself, and which I have already indicated. Here arises the question whether the poison which excites erysipelas is always the same, whether it is specific. This cannot be accurately answered: *in its favor* is the fact that the form of the cutaneous inflammation induced is always the same, although varying in intensity and extent; *against* it we may say that erysipelas is probably caused by various kinds of putrefaction, by miasma, perhaps also by some animal poisons. Possibly in all of these poisonous substances there might be one certain material which induced erysipelas, particularly a variety of material, which had a specific affinity for the lymphatic vessels of the skin; it must be acknowledged that, under certain circumstances, existing at some particular time, such a material may develop more readily and extensively than at other times. It has often been asserted, and of late more particularly by *Orth*, that erysipelas extends by micrococcus vegetation from the wound to the skin. Although the spread and reproduction of the erysipelas contagion much resemble those of a ferment, there s as yet no proof that in erysipelas the micrococcus is the bearer of ich a ferment, still less that it is *only* micrococcus. I do not upold the correctness of this view. I have sometimes found coccus d streptococcus in the serum of erysipelas vesicles, but they are o found in blisters from burns or sweating, in small-pox pustules, ; and this is no proof that these diseases are due to micrococcus. It is doubtful whether the suppurations induced in rabbits by inoculation with the serum from erysipelas blisters is identical with the erysipelas of man. The most recent work on erysipelas, by *Lukomsky*, shows the near relation of micrococcus to erysipelas; I can confirm his observations from others made at my clinic by *Ehrlich*; but interesting as these are, they cannot settle the vexed question as to the etiology of erysipelas. The disease always begins with a rapidly-increasing fever, which continues as long as the eruption lasts; it may be either remittent or continued, sometimes terminates with critical symptoms, sometimes gradually. I have no extensive experience of the so-called idiopathic erysipelas capitis et faciei; from what I have seen, it seems to me very probable that this also starts from slight wounds (excoriations on the head or face) or inflammations (nasal catarrh, angina), and is also chiefly of toxic origin.

The *treatment* of erysipelas is chiefly expectant. We may try prophylaxis by carefully cleansing the wound, and thus keeping off every thing that can favor the occurrence of erysipelas; and when several cases occur in hospital, we should carefully guard against too many of them being in one ward, and occasionally some of the wards should be entirely vacated and ventilated for a time, to prevent the development of a more intense erysipelas contagion (little as we certainly know of it).

As to the local treatment, a series of remedies has been tried to prevent the advance of the erysipelatous inflammation and arrest the disease at its commencement. For this purpose we circumscribe the borders with a stick of moist nitrate of silver or with strong tincture of iodine. According to my experience, this does little good, so that of late I have entirely left off this treatment. Older physicians thought that cold might force the cutaneous inflammation back, and thus greatly favor inflammation of the internal organs. Although this cannot be regarded as proved, a series of facts renders the use of cold apparently unadvisable. We have already mentioned that the occasionally great œdema may induce gangrene, which of course would be greatly favored by intense cold; and the application of bladders of ice to a large surface, as to the back or the whole face, is scarcely practicable; lastly, the ice does no good, as in spite of it the disease runs its typical course, for here almost more than in any other inflammation the local process and general infection go hand in hand. In the affected skin the patient has a disagreeable tension, a slight burning, as well as great sensitiveness to draughts or other changes of temperature. Hence it is advisable to cover the diseased skin and protect it from the air. This may be done in various ways:

the simplest, which I usually employ, is to smear the surface with oil and apply wadding; the patients are generally satisfied with this. Others sprinkle the inflamed skin with flour or powder, or scatter finely-rubbed camphor in the wadding that is to be applied, thinking thus to act specially on the local process. If vesicles form, they should be opened with fine needle-punctures, and the loosened epidermis be left to dry. If gangrene develop anywhere, moist warmth in the form of fomentations or poultices should be applied till the eschar has detached and healthy suppuration begun, which is then favored by dressings of charpie dipped in chlorine-water. If, after erysipelas, abscesses form in the subcutaneous tissue, they should be opened early and treated like any suppurating wound.

Among the internal remedies, we have one which may perhaps arrest the development of some cases of the disease. If in strong, otherwise healthy persons, in whom the gastric symptoms are very prominent, we give an emetic, the advance of the erysipelas is often checked. This is not absolutely reliable, but you may try it in suitable cases. Subsequently you employ only the ordinary cooling remedies. If symptoms of debility show themselves and the disease drag on, you should begin with tonics and stimulants; you may daily give a few grains of camphor or quinine, or some wine.

The inflammations of internal organs occasionally complicating erysipelas are to be treated *lege artis*, and in meningitis you must not be afraid to keep a bladder of ice constantly on the head, even if the scalp is affected by the erysipelatous inflammation.

4. *Inflammation of the lymphatic vessels* (*lymphangitis*), actual inflammation of the lymphatic vessels, occasionally occurs in the extremities under various circumstances, which will be mentioned immediately. The symptoms, in the arm for instance, are as follows: There is a wound of the hand; the whole arm becomes painful, especially on motion; the axillary glands swell and are sensitive, even on the slightest touch. If we inspect the arm carefully, we find red striæ, especially on the flexor side, running longitudinally from the wound toward the glands; these reddened portions of skin are very sensitive. At the same time there is fever, often a coated tongue, nausea, loss of appetite, and general depression. The termination may be in one of two directions: under proper care and treatment, there is generally resolution of the inflammation; the striæ gradually disappear, as do also the swelling and pain of the axillary glands; the fever ceases at the same time. In other cases there is suppuration; the skin of the arm reddens gradually and extensively in a few days and becomes œdematous. The swelling of the axillary glands increases, the fever becomes greater, and there may even be chills. In a few

days fluctuation occurs most frequently in the axilla, occasionally elsewhere in the arm, the abscess opens spontaneously or is incised, and pus, such as is usually contained in a circumscribed abscess, is evacuated. Then the fever subsides, as do also the pain and swelling; and the patient speedily recovers from his disease, which is often very painful and troublesome. The termination is not always so favorable; but, in lymphangitis from poisoned wounds, pyæmia is occasionally developed, in the subacute form most frequently; of this more hereafter. In one case with lymphangitis of the leg, where the patient had chronic inflammation of the kidneys at the same time, I saw the inguinal glands with the superjacent skin become gangrenous, after they had been enormously swollen. This termination is very rare, although the pus in these inflammations of the lymphatic vessels, especially after poisoning with cadaveric matter, is occasionally putrid in character. Acute inflammation of the lymphatic *glands*, terminating in resolution or suppuration, occurs as an idiopathic disease; in such cases we cannot see the connection, by red lines along the lymphatics, between a wound, or another point of inflammation, and the lymphatic glands; this may be because only the superficial vessels appear as red cords in the skin, while the deeper ones, even when inflamed, are not recognizable to the sight or touch. Hence in the patient we only know superficial lymphangitis. One of the peculiarities of this disease is, that when it occurs in the extremities it rarely extends beyond the axillary or inguinal glands. Once in a case of lymphangitis of the arm and adenitis of the axilla I saw pleurisy occur on the same side, which possibly may have resulted from extension of the inflammation through the lymphatic vessels.

We know very little of the pathological anatomy of lymphangitis of the subcutaneous tissue, scarcely more than we can see with the naked eye on the patient, for this disease is scarcely ever fatal when it only attacks the lymphatic vessels, and in animals it can only be very imperfectly induced by experiment. The cellular tissue immediately around the lymphatic vessels is decidedly implicated, the capillaries dilated and distended with blood. We cannot decide whether the lymphatic vessel is obstructed in the later stages by coagulating lymph, or whether coagula form in the lymph at the start and irritate the walls of the vessels. If we may transfer the observations on uterine lymphangitis, which so often occurs in puerperal fever, to the skin, in certain stages there is pure pus in the dilated lymphatic vessels; the vicinity of these vessels is infiltrated with serum and plastic matter; the plastic infiltration of the cellular tissue increases to suppurative infiltration, or even to formation of abscess, in which the thin-walled lymphatic vessels themselves disappear; the finer the net-work of lymphatic vessels, the more difficult it is to distinguish lymphangitis from inflammation of the cellular tissue. From the illustrations of *Cruveilhier* (Atlas, Livre 13, Pl. 2 and 3), we may derive an idea of puerperal lymphangitis, and carry this to the same affections in other parts. The red striæ that we see in the skin can only be caused by dilatation of the blood-vessels around the lymphatics, not by blood forcing its way into the latter; hence in patients we really see the symptoms of perilymphangitis induced by contact with the poison streaming in the lymphatic vessels. We know the changes in the lymphatic glands rather better. In them the vessels are much distended, and the whole tissue greatly infiltrated with serum; quantities of cells fill the alveoli tensely, which probably at first impedes and finally arrests altogether the movement of the lymph in the gland; this blocking up of the gland will to some extent prevent the extension of the morbid process.

Lymphangitis may occur in any wound or point of inflammation; but in my opinion it is always the result of irrritation from a poison passing through the lymphatic vessels. The nature of this poison may vary; it may be decomposed secretion from a wound, putrid matters of all sorts (especially that from the cadaver), or matters which from excessive irritation form an inflamed point. We have already stated that the friction from a boot-nail may excite a simple excoriation into a diffuse inflammation, in which a (phlogistic) poison may and often does form, and excites lymphangitis; the same thing occurs in points of inflammation from other causes; by increased irritation a material is formed in the inflammatory focus itself, which proves very irritant to the lymphatic vessels and their surroundings; even a poison encapsulated in an inflamed part may by increased pressure of the blood be driven into the lymphatic vessels, and thence into the blood, although without this cause it might have remained quiet, and been gradually thrown off or eliminated by suppuration. The following case may serve as an illustration: One of my colleagues had a slight inflammation on the finger, from a dissecting wound; this inflammation was purely local, scarcely observable; on a short trip in the Alps he became heated, in the evening he had a lymphangitis of the arm and high fever; the active movement and consequently increased action of the heart had driven the poison, previously lying quiet in the circumscribed point of inflammation, through the lymphatic vessels into the blood.

[Still more peculiar are the cases where a lymphangitis starts from a part which has been inflamed and recovered; we must assume that in such a cicatrix, as in the lymphatic glands, a poisonous substance may remain latent for a time, and then from some irritation, or from a greater flow of blood (from exercise, overheating, etc.), the poison is carried into the circulation, and we have acute lymphangitis, often accompanied by severe phlegmonous inflammation.

Hence it is proper when patients have suffered from local suppuration, especially if caused by infection, even after it has healed, for them to avoid for a long time all muscular strain, heating, or anything that may hasten circulation of the blood or lymph.]

Why, in the different cases, we have sometimes diffuse phlegmonous inflammation, sometimes erysipelas or lymphangitis, cannot be certainly stated, though it *may* be due to purely local causes, and to the character of the poison.

[Some surgeons regard erysipelas, lymphangitis, progressive phlegmon, acute septic œdema, and the analogous inflammations of internal organs, as identical; *Virchow* was of this opinion, and the same view is now held by *Tillmanns.* But, in spite of their occurring in combination, I would regard them as clinically distinct, and when they occur together one may recede as the other advances. Local perilymphangitis may possibly be due to pus-cells from some point of inflammation escaping through the lymphatic vessels.]

From our present knowledge of the passage of cells out of the vessels we may imagine that pus-cells developed in the wound thence pass into the lymphatic vessels, wander through the walls of these vessels, and as bearers of an irritating substance excite perilymphangitis, while the cells, flowing more rapidly in the centre of the vessel, enter the blood, and thus perhaps induce fever before the local disease has attained any considerable extent.

The object of *treatment* in recent cases of lymphangitis is to obtain resolution if possible, and to prevent suppuration. The patient should keep the affected limb as quiet as possible; should there be gastric derangement, an emetic is very beneficial. The disease not unfrequently subsides after the purgation and sweating induced by the emetic. Among the local remedies, rubbing the whole limb with mercurial ointment is particularly efficacious; then the part should be covered warmly so as to maintain an elevated, regular temperature. For this purpose we may employ wadding or moist warmth. Should the inflammation increase in spite of this treatment, and diffuse redness and swelling occur, suppuration will take place at some spot. This diffuse inflammation is no longer limited to the lymphatic vessels, but the entire subcutaneous tissue participates in it more or less. As soon as fluctuation is distinctly perceived, an opening should be made, and the pus evacuated. Should healing be retarded, it may be hastened by daily warm baths; these are particularly useful where there is a great tendency for the disease to return to a spot once attacked. A septic poison encapsulated in the lymphatic glands, if forced into the circulation by fluxion to the glands, may induce new lymphangitis and phlegmonous periadenitis; this explains the repeated relapses, and the latency of the disease after infection, especially in dissecting wounds.

[When speaking of injuries it was stated that the injury itself caused no inflammation, but that some complication must be superadded to excite traumatic inflammation; the most frequent of these complications have also been described. Strictly speaking, inflammation after injuries is an accidental traumatic disease, standing between so-called normal traumatic inflammation and accidental traumatic diseases. But it is customary to regard those cases of inflammation following an injury as normal if the local symptoms remain limited to the injured parts. This view is doubtless because in the great majority of cases left to themselves, some irritation always occurs, not from the injury, but from necessary complications. When, in following chapters, we treat of accidental traumatic diseases, we do not mean so-called traumatic inflammation. I must now inform you that occasionally other peculiar inflammatory and gangrenous processes affect the wound, and may induce severe and usually feverish constitutional diseases; some of these may also come without anything unusual having been noticed about the wound. Lastly, in an existing wound, or at the time of its reception, as in a bite from a poisonous or diseased animal, substances may enter that may cause local inflammation or general blood-poisoning. Of these we shall now treat, and will first speak of the local symptoms that may accompany a point of inflammation.

Recent wounds may be infected by decomposing secretion entering the open spaces of the tissue, or by infected objects, such as unclean sponges; these contain vegetations or germs of micrococci, which, being introduced into the wound, there find conditions favorable for their development, and spread; and, wherever they reach, induce progressive inflammations.

Among the differences between diphtheria of wounds and hospital gangrene are the progressive pulpous destruction of the edges of the wounds in the former, the great sensitiveness of the granulations, and their tendency to bleed. But I believe, with *Heine*, who has written an excellent monograph on hospital gangrene, that the variations in the course of this and diphtheria of wounds is not due to any essential conditions. There are various grades of wound diphtheria, whose common sign is infiltration of the granulations by rapidly-hardening exudation, which is at once followed by its gangrene. Sometimes the diphtheritic infiltration affects at the same time the surface and edges of the wound, and progresses so rapidly as to destroy a considerable layer of tissue in a few hours. This forms the pulp that may be rubbed off, thereby exposing bleeding, ragged, deep tissues. *Heine* has claimed that hospital gangrene may result from infecting wounds with the virus of true diphtheria of mucous membranes, and that he has seen patients attacked with pharyngeal diphtheria after exposure to hospital gangrene. Moreover, the paralyses peculiar to diphtheria are said to accompany hospital gangrene. It seems as if both diseases were especially malignant in certain epidemics.

The most important treatment for diphtheria of wounds is prophylactic, and consists chiefly in the application of *Lister's* dressing whenever possible, and closely watching insignificant injuries which cause infection because they are not cared for.

For local application, a most excellent remedy is iodoform; the diseased tissue may be scraped out, and the powder freely applied. It is especially useful in cases where the mouth is affected, or where operations have been done on the jaws.

The sensitiveness to pain of the reddened skin gives a tolerably certain diagnostic symptom for distinguishing erysipelas from simple erythema, or from so-called pseudo-erysipelas or phlegmonous inflammation. In erysipelas, light pressure or stroking the reddened skin with the finger is painful, while it is not so in erythema; and in phlegmon, hard pressure causes a dull, deep pain.

On carefully examining patients with so-called spontaneous or non-traumatic erysipelas, you may almost always find a small skin-wound, or, in old persons with erysipelas about the nose, there may be an abscess about a hair-follicle in the nostril; there will probably be some sore of which the patient was unconscious. As these points are often scratched with dirty finger-nails, they might very easily be infected. Hence, undoubtedly, most so-called spontaneous cases of erysipelas are truly traumatic. *Tillmanns's* inoculations of rabbits seem to have induced true erysipelas, as shown by the temperature and examination of the animals experimented on. He observed that the presence of coccus vegetations was not essential for the erysipelatous process; also that the transfer of erysipelas by subcutaneous injection of serum from erysipelas vesicles was possible, but did not always succeed; and, lastly, in successful inoculations, the fluid always contained cocci, although fluid containing them did not always cause erysipelas. His experiments show at there may be erysipelas with or without fungi, and that the ison may be transferred by fluid with or without cocci. Accordg to his views, erysipelas poison may also enter the body by solurns of continuity of the respiratory or digestive tracts.

In the treatment of erysipelas, *Hueter* recommended very highly bcutaneous injections of two-per-cent. solution of carbolic acid, t they are only of use at the very onset; a *Pravaz's* syringel of the solution is scarcely enough for a space half as large as a rd.

Internally, patients stand large amounts of stimulants, and they ould have good, easily-digested food.]

## LECTURE XXV.

5. Phlebitis; Thrombosis; Embolism.—Causes of Venous Thrombosis; Various Metamorphoses of the Thrombus.—Embolism.—Red Infarction, Embolic Metastatic Abscesses.—Treatment.

5. *Phlebitis; Thrombosis; Embolism; Embolic Metastatic Abscesses.*—Besides the above forms of inflammation, there is often another phlebitis and thrombosis, which, starting from a wound or point of inflammation, is at first local, but afterward spreads in a peculiar manner to several organs. In persons dying from this disease we find pus, or friable, purulent, or putrid clots, in the thickened or partly-suppurating veins near the injured part. Often, also, there are abscesses in the lungs, more rarely in the liver, spleen, and kidneys. *Cruveilhier* proved that these metastatic abscesses were connected with the pus in the veins; but the mode of this connection was not explained till subsequently.

What I shall tell you to-day on this subject is the result of numerous investigations and experiments, for which we are indebted to *Virchow*, and which have been so often repeated and confirmed by different persons that there can be no doubt of their correctness; I have myself studied the subject a good deal, and shall at the proper places state where I have arrived at different results. It would lead me too far to follow this great work of *Virchow* historically, and to give you an epitome of it; I must leave it to your own industry to study these works, and content myself with giving you a short *résumé* of the positive results.

The first important question is, What is the relation of the coagulation of the blood to the inflammation of the vessel? The former view, that the coagulation is due to the inflammation of the wall of the vessel, is purely hypothetical, and not susceptible of proof. On the contrary, we know from the investigations as to the formation of thrombus after ligation of arteries, and of the process of healing of injured veins, that there is immediate coagulation of blood in the injured vessel, before there can be any inflammation of the walls of the vessel. The blood-clot forming in veins after their injury, and constituting their thrombus, is usually short, it is true, but we may readily imagine that it should increase in size from continued deposits of fibrine. You know, from your studies in physiology, that we cause coagulation of the fibrine by whipping the blood. During the motion of the blood the coagulating fibrine deposits like crystals on a rough body, and you can readily satisfy yourselves experimentally that such a body, as a cotton thread, introduced into the vein of a living animal, soon becomes covered with fibrine. Thus roughnesses of various kinds in the vessels may give rise to more or less extensive coagulations of the blood. These roughnesses may certainly form on the inner wall of the vein as a result of inflammation, and coagulation of the blood may thus be induced. Projections into the calibre of the veins may be caused by small abscesses in the walls; formerly, it was supposed that there was a fibrinous coagulation on the inner surface of the inflamed vein, as on an inflamed pleura; it can scarcely be decided whether this really occurs; what was formerly considered as such has been found to be a discolored peripheral layer of the blood-clot.

Besides the mechanical cause of the compression, which favors coagulation, in inflammation of any tissue there is another factor having the same effect, namely, the changes in the intima of the vessels, especially of the veins. If we do not know the positive chemical conditions under which the blood in the vessels must coagulate, since the classical investigations of *Brücke* we do know that the normal living intima of the vessels has the special property of keeping the blood fluid, and that coagulation occurs when the intima loses its normal qualities. But in the veins, as in the capillary walls, it loses its normal qualities through inflammation, as is shown by the more recent investigations (*see* Lecture XXII.); these show, indeed, that the inflammatory alteration of the walls of the vessel does not of itself at first induce either complete stasis or thrombosis; however, it is not improbable that the latter is at least favored by the alteration of the walls of the vessel. Hence, the recent views on inflammation would, in some cases at least, confirm the old view that inflammation of the walls of the veins may cause thrombosis (even without leading to abscess in their walls); but further investigations in this direction are desirable. Clinical observations also speak in favor of such a course; for it has been proved that periphlebitis (analogous to perilymphangitis) often precedes phlebitis and thrombosis.

At all events, inflammation of the walls of the vessel does not always cause the coagulation; much more frequently the clot forming in a vessel after injury, under certain not accurately-known circumstances, forms the starting-point for further coagulation, and finally for inflammation of the wall of the vessel. Besides injuries, there is a second factor from which coagulations may result, viz., from retardation of the current of the blood from friction, as in contraction of the vessel; this variety may be called *thrombus from compression.* It also is independent of inflammation of the wall of the vein, but may result from inflammation of the perivenous tissue; for in severe inflammation a tissue, especially when it is under the pressure of a fascia, may swell so much, partly from serous, partly from plastic infiltration, that the vessels will be compressed, and stasis and coagulation of the blood be thus induced. These thrombi, from compression in very acute inflammation, and especially in acute accidental inflammation of cellular tissue around wounds, are more frequent than primary traumatic thrombi; it is the most dangerous variety of thrombus, as it is most liable to puriform deliquescence. In rapid dilatation of a vessel, also, according to physical laws, the current of blood is much retarded; then coagulation takes place at the point of dilatation, as we shall hereafter see in aneurisms and varices; these are called *thrombi from dilatation.* Furthermore, the current of blood may be retarded from insufficient contraction of the heart and arteries; as this occurs chiefly in persons debilitated by age or severe exhausting diseases, it is called *marasmic thrombus.* This, also, is evidently independent of inflammation of the veins, and occurs most frequently in parts distant from the heart.

You must remember that in all these cases the thrombi are at first small, and gradually grow from deposit of more fibrine. It has not been proved that, in cases where the thrombus attains a considerable extent, there is any abnormal increase of fibrine in the blood, although this might be supposed. *Why* traumatic thrombi should extend so far in some cases of injuries of the veins, we can only understand in cases where extensive ruptures of the veins are caused by extensive contusions, and extensive disturbance of the circulation is thus induced. But, in cases where a widely-branched thrombus results from a punctured or incised wound of a vein (as from venesection), it is often difficult to explain the cause without resorting to disputed hypotheses. Thrombi from injury and compression, and their sequelæ, particularly claim our attention, while those from dilatation and marasmus we rarely meet in surgical cases. It has been observed that venous thrombi ending in suppuration are far more frequent in hospitals than in private practice, and this tendency to coagulation of the blood has been referred to the hospital atmosphere and the miasma it contains. That hospital miasm (itself a very indefinite and very variable thing) should directly induce coagulation of the blood, can neither be proved nor denied. According to my idea, the connection is probably only indirect: toxic-miasmatic infection of a wound, whether induced by instruments, dressings, or otherwise, as previously stated, excites acute suppurative inflammations around the wound, sometimes as ordinary cellular inflammation, sometimes as diffuse lymphangitis, etc.; thrombi from compression are caused by these inflammations, just as happens in acute phlegmonous inflammation outside of the hospital; hence the influence of miasmatic poisoning in inducing venous thrombosis is not direct, but indirect, acting through the inflammation.

The next question is, What becomes of the blood coagulated in the vessels, and what is its relation to the wall of the vessel? From the injuries of arteries and veins, we are only acquainted with one metamorphosis of the thrombus, namely, its organization to connective tissue. In extensive venous thrombi this is a great rarity, and leads of course to complete obliteration of the vein. Let us take a very simple case, a venesection thrombus. After a bleeding, say from the median vein, from an acute inflammation of the cellular tissue there is a coagulation of blood in this vein, and also in the cephalic and basilic veins, down to the wrist and up to the axilla. From the disturbance of the circulation thus caused, there is great œdema of the whole arm; when this subsides, we may distinctly feel the subcutaneous veins as hard cords. The course may vary: first, the affection may possibly end in resolution—under timely treatment this is usual; the patient should be kept in bed, as he is usually feverish; the arm should be kept absolutely quiet, and covered with a compress thickly coated with mercurial ointment. At the same time we give a purgative, and, if the tongue be coated, an emetic. Under this treatment, the swelling of the arm usually decreases, and the fever subsides. Then the firm venous cords can be plainly felt; in six or eight days they become softer, and finally cease to be perceptible. We very rarely have the chance to examine such cases anatomically in the early stages. Hence, we cannot decide to what extent, if at all, the walls of the vein participate in this coagulation of the blood; but, from the symptoms and the examination of the patient, it would appear that the fibrine coagulated in the vessels is gradually reabsorbed and mingles with the blood without injury, like other blood that has been diffusely extravasated in the tissue. The second termination of inflammation of the arm after venesection, complicating thrombosis, is the formation of abscess. The first symptoms are those above described; but then, either in the bend of the elbow, the arm, or the forearm, a more circumscribed inflammatory tumor forms; this increases gradually, and finally fluctuates distinctly. On incision, pus is evacuated from a larger or smaller cavity, the swelling of the arm then gradually de creases, the abscess heals, and complete cure may result. Anatomical examination of these cases shows that there has been suppurative inflammation in the connective tissue around the vein. We also find that the coats of the thrombosed veins are greatly thickened; this is to be regarded as a result, not as a cause of the thrombosis. I will here add that the diagnosis of a venous thrombus cannot always be made, from the vein feeling like a hard cord; for occasionally inflammation in the cellular tissue around the vein may extend, and cause condensation and tube-like thickening of the sheath of the vessel, which may readily cause it to be mistaken for thrombus, though it does not necessarily lead to it. I have twice seen this mistake of periphlebitic cellular induration for thrombus of the saphenous vein, and I consider it impossible to make a certain diagnosis in all cases. The fact that such a periphlebitis, which is perfectly analogous to perilymphangitis, and in which the walls of the veins certainly participate, can exist without thrombosis, proves beyond a doubt that the latter is not necessarily the cause of inflammation of the veins, as was formerly supposed. Another possible metamorphosis of thrombus is friable disintegration. In this, softening of the clot usually begins at the point where the thrombus began, that is, at the oldest part. The fibrine breaks down into a pulp, which is yellowish or brownish, and smeary in proportion to the number of red blood-corpuscles contained in the coagulum. This disintegration spreads more and more; even the tunica intima of the vein does not escape, it becomes wrinkled and thickened. The thrombus changes to pus, which mingles with the detritus of the fibrine, while the walls of the veins and surrounding cellular tissue are greatly thickened; occasionally, although rarely, small abscesses form in the walls of the vein. Hence, here the inflammation of the wall of the vein is to be regarded as the result of softening of the thrombus, and the pus which we then find in the vein does not come from the wound (the old idea), but forms in the vein from the blood-clot. Often, also, the puriform fluid is only fluid fibrinous detritus, while in many cases good thick pus, with fully-developed corpuscles, may be found in these veins. If the wound be putrid, the fibrinous detritus in the vein may also assume a putrid character, putrid fluid being taken up by capillary action of the thrombus from the wound and acting as a ferment on the disintegrated fibrine. This capillary action of the thrombus might also be supposed to cause an action of the decomposed secretion on the blood. Of course there can be no extensive flow of pus or other secretion from th wound into the vein, as the opening in the vessel is plugged by t thrombus. Should there be a rapid disintegration of the venous thro bus from the peripheral to the central ends, which is rare, there wo at once be venous hæmorrhage, and the formation of a new thrombus, so that even then there could be no entrance of the pus from the wound into the vein, or of that from the vein into the blood; moreover, the pus forming and collected in the vein is so shut off by the central end of the thrombus, that it cannot mingle with the blood; at least this could only happen if the central end of the thrombus should be entirely broken down, but this probably happens very exceptionally, for in most cases there are constantly new deposits of fibrine, while disintegration goes on from the oldest parts of the thrombus. You will thus understand that the entrance of pus into the injured vein cannot readily occur, but that, as will be soon stated, the circumstances must be very peculiar to render this possible. I must here briefly interrupt the description, to state that *Virchow* does not distinctly acknowledge the transformation of the thrombus to pus; I have no doubt on this point: if the blood-cells in the thrombus have the power of increasing and changing to tissue, as seems most probable, there is no reason for not referring to them the formation of pus in the thrombus, just as we do to the white cells wandering out of the vessels, for the coagulation of the blood is not firm enough to entirely prevent cell-movement. That the thrombus may change to true pus by division of the white blood-cells does not appear to me disproved; we have already mentioned that this pus, which is usually encapsulated, does not enter the circulation, or does so very rarely, and hence has no direct connection with pyæmia. To resume my experiences of venous thrombi, and the history of thrombus, they are to the effect that most venous thrombi are the result of very acute inflammation of cellular tissue, (especially under fasciæ, or tense skin, and in bone), and that the coagulum undergoes the same metamorphoses as the inflammatory new formation. If the latter lead to formation of tissue, the thrombi are also organized to connective tissue; if the inflammation goes on to suppuration or putrefaction, the thrombi also suppurate or putrefy and break down. This is the easier to understand, as we know, from *Von Recklinghausen's* and *Bubnoff's* investigations, that the cells from the tissue may pass through the walls of the vein into the thrombus. The walls of the vein have the same fate as the thrombus and surrounding tissue: they are infiltrated with plastic matter, and become thicker, or they suppurate.

Thrombus, with phlebitis, may also run its course as a purely local disease, as not unfrequently happens after venesection, and in some other cases. Then there can only be further danger when the thrombus is friable, or when there is purulent or putrid destruction of the coagulum. The central end of the thrombus (as we stated when speaking of arterial thrombus) usually extends to the point where the next branch joins, and has a conical end, which projects a little (Fig. 67, $a$), and, if the coagulum loses its firmness, a portion of the coagulum may be torn off by the current of blood, and pass into the circulation; this passes into the larger veins, thence into the right heart, thence to the pulmonary artery, in whose branches it is finally arrested at some point of bifurcation, as its size does not allow it to pass farther. This branch of the pulmonary artery is now closed by a clot of fibrine, as by a cork, a so-called *embolus;* the immediate consequence is a lack of blood in the parts of the lung previously supplied by the plugged artery. This local lack of blood (ischæmia of *Virchow*) does not usually last long, but blood enters the empty artery from small collateral arteries; it is true, blood may thus again enter the vein, but it comes from the small collateral branches, and flows very slowly, and may at last stop altogether, and coagulation extend backward through the capillaries even into the thrombosed arterial branch. Thus, as a result of embolus in the artery, the whole corresponding vascular territory is thrombosed; there may also be ruptures of the vessels, hæmorrhages; as the arteries of the lungs, spleen, and kidneys, constantly divide into smaller branches, and thus the vascular territory constantly enlarges toward the periphery, and resembles a cone with the apex in the organ, so the part in which the above coagulation occurs must be shaped like a wedge or cone. In pathological anatomy these coagulations due to embolism have been called "red or hæmorrhagic wedge-shaped infarctions." Frequently as these wedge-shaped infarctions occur, they are not a necessary result of embolism; for, when the arterial collateral circulation is strong enough in the ischemic part to drive the blood through the capillaries, as is the case in otherwise healthy persons and in animals, as well as in emboli causing little mechanical or chemical irritation of the tissue, there is no infarction, at all events no considerable disturbance of circulation, but we have simply to consider the local processes around the embolus, as foreign bodies in the branch of the artery. These local processes depend on the character of the embolus; if the latter be a pure fibrinous clot, there is a slight thickening of the wall of the vessel at the point where the embolus is

FIG. 67.

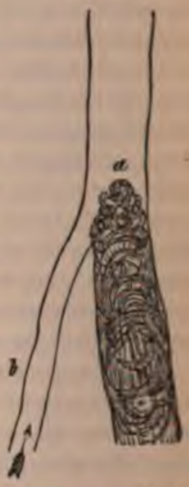


Diagram: $a$, central end of a venous thrombus projecting into a larger trunk; $b$, a branch without thrombus; the blood flowing through it may detach and carry into the circulation the end of the thrombus $a$.

located (usually where the artery divides into smaller branches), and the latter may have new clots deposited around it, and be organized to connective tissue, or be reabsorbed. Should the embolus consist of a fibrinous clot impregnated with pus or putrid matter, it excites suppurative or putrefactive inflammation, not only in the wall of the vessel, but also in the parts around. The metamorphosis of the red infarction in part depends on its size, partly on the grade of the circulation still continuing in parts of it, and partly on the embolus causing the trouble. If the latter be innocuous and the infarction be small, or if it be still nourished by some vessels not thrombosed, the coagulum forming the infarction may again be dissolved, or else become organized to a connective-tissue cicatrix. If the embolus be innocuous, but the thrombus extending completely through the whole infarction, the tissue and coagulum slowly disintegrate to a yellow, granular, dry pulp, which becomes encapsulated, and may calcify; this is *yellow dry infarction.* If the embolus be impregnated with putrid matter or pus, it excites putrid or suppurative inflammation all about it; the infarction also becomes putrid or purulent, and abscesses form. As we were just speaking of the lungs, we may here mention that these abscesses, which are usually peripheral, often excite pleurisy; that they are most frequently multiple in both lungs, and may even induce suppuration of the pulmonary pleura over the abscess, and may thus occasionally cause pneumothorax.

You can hardly imagine, gentlemen, what labor it costs to demonstrate this connection between venous thrombi and abscess of the lung, so that I can here announce it to you as a simple fact. You will read the classical works of *Virchow*, *Panum*, *O. Weber*, and others, on this subject, with astonishment; it would take too long for me to enter into the subject more fully; we shall here assume the right of only taking the facts from these works. We now understand lung infarctions and abscesses; but how is it with those that occur under like circumstances, although much more rarely, in the liver, spleen, kidneys, and muscles; are these also always dependent on emboli? A few years since we could not have answered this question with certainty; now we may affirm it. From experimental investigations, especially those of *O. Weber*, it is established that certain forms of emboli, especially flocculi of pus, pass the pulmonary capillaries without difficulty, may enter the left heart, and thence the systemic circulation, and be arrested in the spleen, liver, kidneys, or elsewhere, and cause abscesses. This explains the rare cases where, with venous thrombus, there are no abscesses in the lungs, while they exist in other organs. If, with abscesses in the lungs, there are embolic infarctions or abscesses in part supplied by the systemic circulation, they may be attributed to the formation of venous thrombi through the pulmonary abscess; portions from these thrombi pass into the left heart, and thence farther. As regards liver-abscesses, *Busch* has observed that retrograde movements of the blood from the right heart take place in the vena cava, and in this way hepatic emboli may occur.

The embolic origin of *metastatic abscesses* is now so undoubted that, from the existence of one of these, we decide certainly on a venous thrombus undergoing putrid or suppurative liquefaction. The discovery of the connection may be easy in some cases, very difficult in others: very easy in cases of thrombus of large venous trunks, and embolism of branches of the pulmonary artery that may be readily reached with the scissors; very difficult where there is simply coagulation in some small venous net-work (as in phlegmonous inflammation or decubitus) and embolism of capillaries of the lungs, spleen, kidneys, liver, muscles, etc.; still, these latter cases are almost innumerable. On favorable objects (as in cerebral capillaries) it has been proved, beyond a doubt, that capillary emboli exist in some cases; it is also certain that small veins become thrombosed in all suppurative inflammations; it is very difficult, often impossible, to demonstrate this anatomically in every case. From what symptoms we conclude whether a coagulum is old or recent, will be taught you in the lectures on pathological anatomy.

Then you will also be told how to distinguish small lobular infiltrations of the lung, such as occur in purulent bronchitis, from metastatic abscesses. I will merely mention here that where a venous thrombus opens into the wound, it may remain firmly organized while its upper part suppurates, breaks down, and is finally swept into the circulation by the neighboring branches in which the blood circulates; this is the only case where pus from the veins enters the circulation without there having been hæmorrhage. After death we recognize this process by finding fluid blood or fresh post-mortem clots in the thickened veins, whose inner walls are rough from adherent layers of the thrombus; if there has been periphlebitis, and the portion of vein has suppurated, we cannot decide with absolute certainty that there has been a previous suppuration of the thrombus.

Here we are only speaking of metastatic circumscribed inflammations, of infarctions, and abscesses; these alone are connected with venous thrombi and emboli. For diffuse metastatic inflammations another explanation must be sought; we shall treat of this more under septicæmia and pyæmia. Nor shall we here discuss the question of fever in phlebitis and in the formation of metastatic abscesses. As phlebitis, with its results, so very often comes as an addition to already-existing acute inflammations, it is difficult to judge how far it of itself excites fever; metastatic abscesses, like all other points of inflammation, undoubtedly induce fever; we should scarcely expect fever from a simple thrombus of the vessels.

In dogs, by inducing numerous small emboli in the lungs by injecting flour or powdered coal into the jugular vein, we may, it is true, excite fever, as was shown by *Bergmann*, *Stricker*, and *Albert;* but this does not always occur in embolism in other vascular tracts, and possibly depends on increased action of the respiratory muscles.

The *treatment* of phlebitis and thrombus is the same as that of lymphangitis and other similar acute inflammations. Careful frictions with mercurial ointment, or, if we fear detachment of the coagulum, covering the part with compresses smeared with mercurial ointment, or with bladders of ice, and absolute rest of the affected part, are indicated. Under pyæmia we shall speak of the diagnosis and treatment of metastatic abscesses. If phlebitis and thrombosis cause local suppuration, the abscesses should be opened as soon as recognized.

[As a result of embolus there is a faulty circle, the inflammatory changes in the walls of the vessels favor stasis, and this again reacts on the intima and arrests its physiological action. When the intima is altered it favors the escape of red corpuscles; there may even be actual tears in the vessel, hæmorrhages, and on section we find the embolic foci not only distended, but the whole tissue full of blood-corpuscles and coloring-matter.

If a thrombus contain cocci and bacteria, as it does in various infectious processes, fungi will develop in the embolic infarctions and increase in the vessels and tissues.

The part of the vessel thrombosed and the tissue it supplied become necrosed, the fluid part being taken up by neighboring vessels, and, as absence of air and moisture prevent decomposition of the remainder of the necrosed part, it undergoes molecular disintegration into pigment, cholesterine crystals, etc., which remain enclosed in a connective-tissue capsule.]

# LECTURE XXVI.

II.—General Accidental Diseases which may accompany Wounds and Local Inflammations. 1. Traumatic and Inflammatory Fever; 2. Septic Fever and Septicæmia; 3. Suppurative Fever and Pyæmia.

## II.—GENERAL ACCIDENTAL DISEASES WHICH MAY ACCOMPANY WOUNDS AND OTHER LOCAL INFLAMMATIONS.

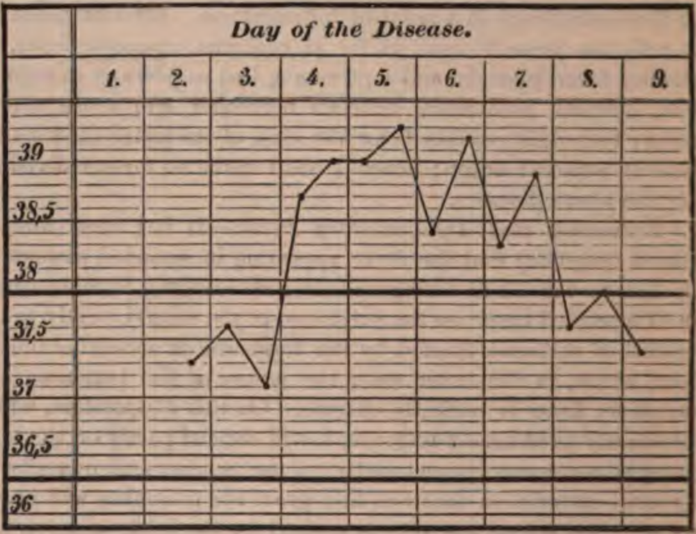
THE local accidental traumatic diseases which we have so far described are always accompanied by constitutional disease, which is chiefly though not always feverish in its nature. Fever is such a complication of symptoms that it may seem very different according to the addition of one or other symptom; now it is generally determined only to say that there is fever when the temperature of the blood is elevated, and to measure the intensity of the fever by the height of the temperature. I do not think it advisable to combat this position, for by abandoning it we should lose the common idea of what we call fever, and throw it back into the old chaos. But I must tell you that there are many and very dangerous general diseases in patients with wounds or other local inflammations, in which no change of temperature of the blood can be discovered; hence the latter is only conditionally a measure of the patient's danger. Besides the elevation of temperature, in fever we have the following chief symptoms: Increased rapidity of cardiac action and respiration, loss of appetite, frequently nausea, feeling of weakness, great sweating, not unfrequently trembling of certain groups of muscles (in chills), more or less mental excitement and blunting of the senses. Fever is a general disease, which may result from many causes; in other words, the number of pyrogenous, like that of phlogogenous substances, is innumerable. According to the quantity and quality of these substances (which we term poisons) that have entered the blood, one or other set of symptoms is more prominent: thus there is fever with very high temperature, while all other symptoms are slight; fever with great blunting of the senses, and but little elevation of bodily temperature; fever whose prominent symptom is severe shivering, so-called chills; fever with disturbance of the gastric functions, fatigue, etc., for the chief symptoms. Why, then, should we not have fever (a state of intoxication caused by materials absorbed from wounds or points of inflammation) with all the symptoms, except elevation of the temperature of the blood? From some cause or other this particular symptom might in some cases be concealed or prevented from appearing. But, as already stated, we shall accept the present view of fever, and only suppose it to exist where we find elevation of temperature of the blood, but must then add that there are cases of severe general, accidental traumatic and inflammatory diseases which run their course without fever.

But there is another common factor of these general diseases that we should bear in mind, viz., that they are all due to reabsorption of matters that form in the wounds or the parts around them, or (what is about the same thing) in a point of inflammation. On this point we agree with the present views, as far as concerns traumatic fever, inflammatory fever, pyæmia, and septicæmia, less so perhaps as regards tetanus, delirium potatorum, delirium nervosum, and acute mania. But many important reasons favor the view of the latter diseases being also of humoral origin; hence I shall make no further divisions among the above diseases.

1. *Traumatic and Inflammatory Fever.*—It has been already explained (page 98) that the fever appearing in wounded patients is partly due to the blood taking up materials resulting from decomposition of mortified tissue on the substance of the wound, partly to the absorption of materials formed by the traumatic or accidental inflammation; hence, in the latter case, the nature of the traumatic and inflammatory fever is perfectly obscure. On this supposition, which we previously tried briefly to prove, it will depend partly on the local advantages for reabsorption, partly on the quality and quantity of pyrogenous material in question, how great the poisoning will prove. There are cases where the vessels opened by the injury close so rapidly, and the whole traumatic inflammation terminates so quickly, that there is no general infection or fever at first, and they may not occur at all; such cases are rare in extensive injuries, they are the ideal of the normal course; in them the plastic infiltration on the edges of the wound leads quickly and throughout the wound to solid organized new formations, growing firmly in the edges of the wound, and passing on to cicatrization immediately or after precedent granulation. If we assume this case as a normal type, every traumatic fever is a pathological accident. We must acknowledge this in theory, but in the great majority of cases, in wounds of any size, fever occurs sooner or later; hence we considered it advisable to treat of traumatic fever in the previous description of the general condition of the wounded patient. We have still, however, to add something to what was then said, which at that time it would have been difficult for you to understand. Let us first speak of the period at which traumatic fever usually appears, and of its course. In many cases, especially where the injury has affected tissues previously healthy, the fever does not begin till the second day, increases rapidly, and, with evening remis-

sions, remains for some days at a certain height, and then cea gradually (rarely within twenty-four hours). According to my v numerous observations, in far the greater majority of cases the tra matic fever begins within two days after the injury. This fever usually represented graphically as follows:

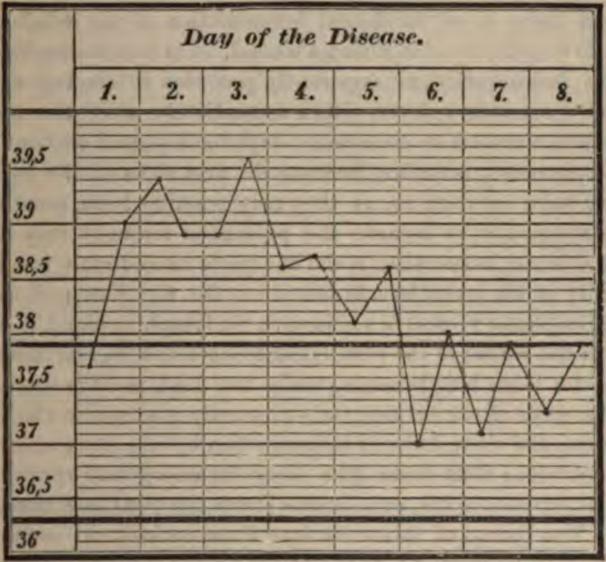
FIG. 68.



Fever-curve after amputation of the arm. Recovery. This and the following fever-curves arranged on the scale of Celsius's thermometer. Each degree is divided into ten pa the horizontal divisions indicate the day of the disease; the curve is made according to morning and evening measurements; the two heavy lines indicate the maximum minimum normal temperature of a healthy person.

The curve shows that, after an amputation of the arm, rendered necessary by an injury (measurement was accidentally neglected the first day), the fever did not begin till the third day, then continued from the fourth to the seventh day; after the eighth day the patient remained free from fever. In other cases, however, secondary fever often occurs immediately after amputation. Such an occurrence of traumatic fever is quite frequent. I explain it as follows: Immediately after the injury the tissue of the edges of the wound was closed by infiltration of plastic matter; the third day this commenced to break down into pus, and to mingle with decomposed shreds of tissue on the surface of the wound, thus inducing a moderately extensive inflammation of the amputation stump, with reabsorption of pus and other products of decomposition and inflammation; this reabsorption goes on till checked by some mechanical cause (diminished pressure, thickening and partial closure of the vessels, etc.). In other cases, the fever begins the very day of the injury; we see this when blood has been enclosed between the flaps of the united wound and it has rapidly decomposed; frequently, also, when operations have been done in tissues infiltrated with the products of chronic infiltration. The following case (Fig. 69) may serve as an illustration of this second class:

FIG. 69.



Fever-curve after resection of a carious wrist, with great infiltration of the soft parts. Recovery.

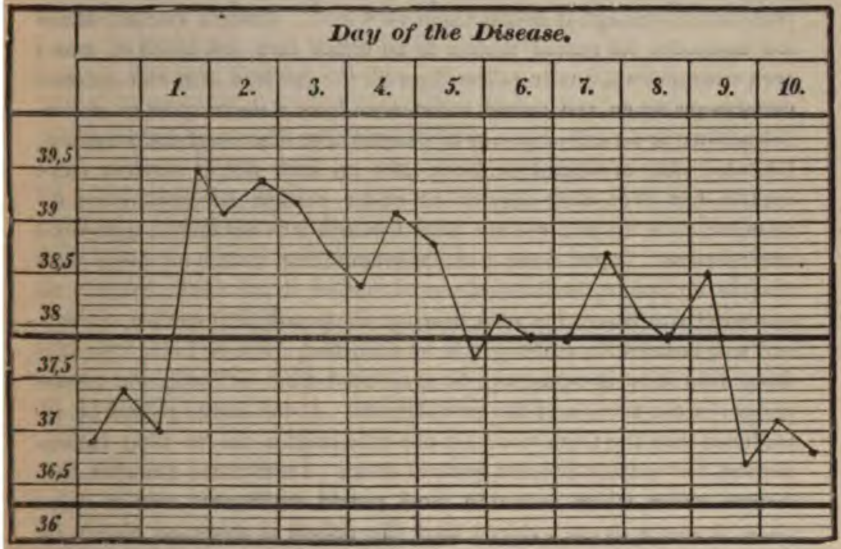
In infiltration of the tissue from chronic inflammation, the finer lymphatic capillaries may be contracted and to some extent closed, and hence, for some time, may not have carried off sufficient serum from the tissue, but the medium-sized lymphatic vessels, like the corresponding veins, which in chronic inflammation have long been exposed to high pressure, are undoubtedly distended, perhaps even gaping, from rigidity of their walls; hence, if not quickly filled with firm plastic infiltration from the start, they take up a good deal of the secretion from the wound; moreover, on the edges of wounds in morbidly-infiltrated tissue, mortification is particularly apt to occur. This explanation of the late and early occurrence of traumatic fever is purely hypothetical; but it is taken from and has been induced by numerous observations. It might also be assumed that in one case the ferment absorbed into the blood acted very slowly, in another very quickly; nothing definite can be said on this point. As I formerly believed that the fever was always caused by nervous irritation, it was necessary to suppose that this irritability was varied, and hence the febrile effect might occur at very different periods, but I have entirely abandoned this theory, without undervaluing the important part played by the nervous system in the origin and symptoms of fever.

Traumatic fever usually lasts a week; it is rarely longer, without some visible local complication.

When there is an accidental inflammation of the cellular tissue, lymphatic vessels, or veins, about a wound, fever occurs simultaneously with this inflammation, or apparently precedes it (coming as an inflammatory secondary fever, either immediately after the traumatic fever or when several or even many days have passed without fever). I say it apparently precedes, because the first signs of the local affection may have escaped us, as they may possibly have presented no sensible symptoms, or because the poisonous material may have infected the blood sooner than it did the parts immediately around; the probability of the latter idea is based on the fact that poison, taken into the lymphatic vessels or veins with the lymph or blood, flows more rapidly in the centre of the vessel than along its walls, and thus quickly reaches the large blood-vessels, while the fluid, moving more slowly along the walls of the vessels, only gradually passes into the perivascular tissue, and there induces inflammation by the phlogogenous poison it contains; thus fever (the blood-infection) may appear before erysipelas, lymphangitis, or phlebitis (from the local infection), is perceived. The course of this secondary fever entirely depends on that of the local inflammation; as the latter begins, the temperature rises rapidly, often with an initial chill. The longer these secondary fevers continue, that is, the longer the poison is kept up, the more dangerous the condition becomes; rapid emaciation, great sweating, sleeplessness, and continued loss of appetite, are bad symptoms; usually in these secondary fevers there is absorption of pus or infection from without. Pronounced erysipelas or inflammation of the lymphatic vessels or glands are the relatively most favorable forms of the accidental inflammations, as sooner or later they generally lead to a certain usually favorable termination, and thus are somewhat typical in their course, although the duration of an erysipelas may vary from three days to three weeks or more, and prove very debilitating; at first the fever-curve rises rapidly, then remains for a time at a certain height, usually with morning remissions; not unfrequently the temperature falls rapidly; the same is true of lymphangitis. Fortunately, it is rare for lymphangitis and erysipelas to extend deep into the cellular tissue and under the fasciæ; in such a case the disease would be classed among the severer inflammations, and would lose its somewhat typical character.

In diffuse, deep inflammation of the cellular tissue, with or without venous thrombosis, the fever does not begin so suddenly, but, from the first, always has a decidedly remittent type, and, like the local affections, is incomputable in its further course; the loss of strength, the emaciation, sweating, sensitiveness, and excitability of the patient, attain the highest grade. Intermittent fever and metastatic inflammations, the chief symptoms of those malignant traumatic fevers which we call "pyæmia," are greatly to be feared in such cases.

Fig. 70.



Fever-curves in erysipelas traumaticum ambulans faciei, capitis et colli, following extirpation of a cancer of the lip. Recovery.

In all these fevers the quantity of urea is increased and exceeds the amount of nitrogenous food consumed; at the same time, according to recent investigations, the weight of the body diminishes considerably.

As long as the constitutional symptoms, especially those due to the fever, do not extend beyond the above, and especially if the disease does not prove fatal, we are generally satisfied with the terms "traumatic, suppurative, or secondary fever." But, if other symptoms occur, and death results, these severer infections have two other names, "septicæmia" and "pyemia." We follow this common classification.

2. *Septic Fever* (*Septicæmia*).—By septicæmia, we understand a constitutional, generally acute disease, which is due to the absorption of various putrid substances into the blood, and it is thought that these act as ferments in the blood, and spoil it so that it cannot fulfil its physiological functions. This disease may be induced in animals by injecting putrid matter into their blood or subcutaneous tissue, and it has been found that large animals (large dogs, horses, etc.) may, under certain circumstances, live through the putrefactive blood-poisoning, although it makes them very sick. Certain circumstances are necessary for putrid matter to be taken into the blood of man; such substances are only taken through the healthy skin and mucous membranes when the putrid substances have a destructive or cauterant action, or an active power of penetrating, like fungi and infusoria. Diseased skin or wound surfaces take up such putrid matters more readily, but even they only do so under certain circumstances; for instance, they do not readily pass through well-organized, uninjured granulations. If we dress a nicely-granulating wound on a dog with charpie dipped in the filthiest putrid matter, if the latter contain no cauterant substance that may destroy the granulation surface, the animal will not sicken, nothing will be absorbed. Hence I conclude that the poison must in some way be prevented from entering the blood-vessels in the surface of the granulations. If the septic poison be introduced into the fresh tissue, it not only excites severe local inflammation, but quickly induces general fever. From these peculiar conditions under which infection from putrid substances usually takes place, it seems to me evident that the poison is absorbed chiefly by the lymphatic vessels, as I have already mentioned. Remember, also, that, in contused wounds, decomposing shreds of firm connective tissue, especially of tendons and fasciæ, often lie for a long time on granulating wounds, without any septic poison passing from them through the superficial vessels of the granulations into the blood; this observation verifies the experiments made on dogs. But, if the poison be not taken up by the blood-vessels, or be taken only under certain circumstances, it is very probable that its absorption is chiefly through the lymphatic vessels. I will not deny that possibly in certain swollen states of the walls of the blood-vessels, as well as from capillary attraction, and also through the thrombi of the vessels, infectious materials *may* reach the blood, nor that cells take up septic molecular substances and *may* wander with them into the blood-vessels; but, on the whole, I consider this mode of infection the exception, especially if the infectious substance be not dissolved, but exist as very fine molecules; if, for instance, it be taken up in the form of dust. Of the healthy parts of the body exposed to the air, it has only been proved

that dust-like bodies (as coal-dust) enter the lungs, and may thence reach the bronchial glands (thence also the blood), while a similar absorption from the walls of the intestines has not yet been observed or experimentally proved. Should the miasmata really be small fungi, that is, molecular bodies, from what has been said, it would seem very probable that the infection may take place through the respiration; if this should be proved, it might be of great practical consequence.

Of late, many attempts have been made to determine what substance in decomposing animal tissue is the true poisonous principle, and for this purpose putrid fluids have been treated chemically till some one body should be found which in the smallest dose should excite the symptoms of septic poisoning. Thus *Bergmann* has produced a body of this nature from decomposing yeast, which he calls *sepsin*. To prove that this body *alone* (whose presence *Fischer* could not prove in decomposing serum or pus) is the poison, it would be necessary to prove the innocuousness of all other bodies chemically formed during putrefaction. But this cannot be done; sulphuretted hydrogen, sulphuret of ammonium, butyric acid, leucin, and some other substances, forming during the putrefaction of organic bodies, also act as septic poisons when injected into the blood; so that I cannot enter into the laborious search for one body in the putrid fluids which shall bear all the blame of the injurious effects. It is very probable that in decomposing fluids, according to their qualities, degree of concentration, temperature, etc., very many different poisonous substances may form, which I further imagine as going on changing till they reach some final terminal stage.

Whether this terminal stage is always the same is still to be determined. This is not the place to discuss such difficult questions exhaustively; so far as my experience, observations, and studies go, I consider it at least probable that the septic matters are formed in the inflamed and gangrenous tissues, and pass to the blood as a developed poison. Opposed to this view is another, that only a ferment goes to the blood from the tissues (ultimately from the air), which soon causes fermentation or decomposition (*O. Weber*). According to this, the absorbed septic matters would not be in themselves poisonous, but would develop poison in the blood from its components.

[Possibly the fever occurring in sensitive persons from constipation may be due to poisonous matters being driven into the absorbents by mechanical pressure. This rise of temperature is rarely over 102°, and is usually only observed in persons under treatment, possibly because they have their temperature taken regularly. Probably putrid gaseous substances are absorbed from the intestinal canal, where they have been under certain pressure. The best proof that the sole cause of the fever was the constipation, is the immediate relief given by an effective enema. The same cause may account for the fever in some strangulated herniæ or acute obstructions of the bowels. If the pressure from without exceed the intra-vascular pressure, the decomposing fluid may be driven into the vessels.

If there be cocci in the septic fluid, there must still be certain favorable conditions to ensure their passing through uninjured granulations; the upper cell layers are constantly thrown off as pus, and the pressure of the blood and lymphatic circulation from below toward the surface interferes with absorption.

Experiments for inducing acute infectious osteomyelitis showed that when the medulla of bones had been injured, feeding the animals on putrid substances sufficed to induce infection of the wound of the bone; so poison must be absorbed from the bowels.

From decomposing macerated meat *Panum* obtained a non-albuminous body, soluble in water, and also a second, very poisonous substance; *Zuelzer* and *Sonnenschein* also discovered a septic substance resembling the alkaloids.

To-day there is little doubt that the impulse to development of the process by which septic matters are formed, is given by a living organized ferment, micro-organisms; and it has been shown that other infectious processes may cause and transfer, by peculiar vegetations, a number of acute and chronic diseases. At present the views appear to be about as follows: Septic or putrid poison does not seem to depend exclusively on microscopic organisms, but to exist in solution in putrid fluids. Non-organized ferments, when kept long at a heat of 160° to 170°, lose their activity, while fluids containing cocci or bacteria under the same conditions retain their septic powers, which are only destroyed by a heat of 220°. If we kill a rabbit by injecting fluid containing bacteria, as *Davaine* and *Stricker* did, and inject some of the blood of this rabbit into a second one, and of this into a third one, it remains fully as active or even more so; by breeding through several generations the virulence of the virus increases. These inoculations do not change the micro-organisms morphologically. Quite recently *Pasteur* has made the important discovery that, as we may strengthen the virus by cultivating it in various generations of animals, so it may be *weakened* by cultivating it in a certain way *outside* of the body. *Pas-*

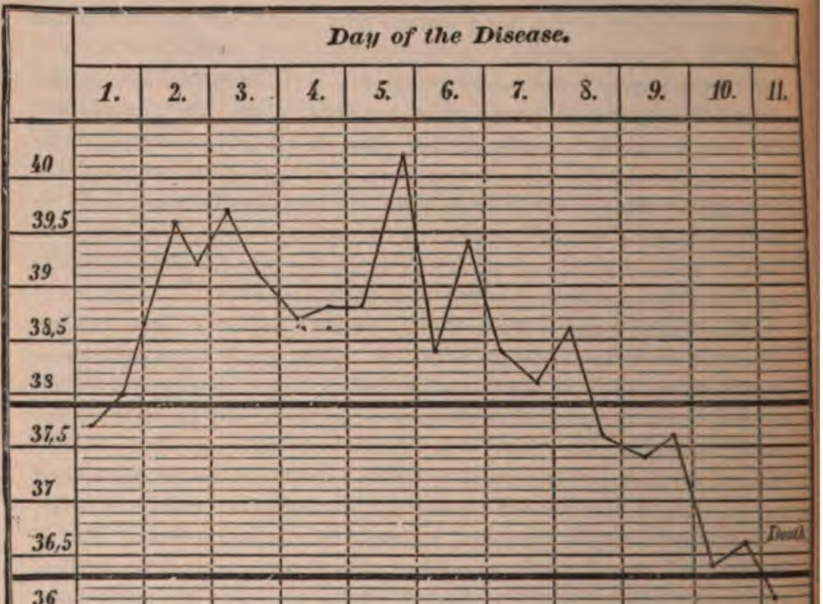
teur verified these observations in so-called chicken-cholera, an infectious disease caused by micro-organisms. He discovered the very important fact that animals which had been inoculated with the weakened virus took the disease, but did not die, and, once having had the disease, were protected against further inoculation with the stronger virus, which would otherwise be fatal. *Pasteur* explains this immunity by saying that the micro-organisms destroyed or changed certain matters in the body of the animal so as to leave no proper nourishment for the development of new micro-organisms. Of course this discovery cannot be transferred, without further investigation, to septic virus.

Fermentation, or the process of breaking up organized bodies into their original non-organic elements, is due to the development and evolution of organized beings, whose species differ with the kind of decomposition or fermentation. These germs come from without, and their development may be prevented by protecting the fermentable substance from contact with the air, or by placing it in air submitted to a temperature that has destroyed the germs which it always contains. Access of the least particle of air not thus treated may induce fermentation. It is claimed that, in the vegetable kingdom, diseases without exception are due to parasites; and that, in the lower animals and man, in all cases where the etiology is *known*, the same holds true. Rabies, glanders, syphilis, variola, and various skin-diseases, we can transfer from one individual to another; measles, scarlatina, whooping-cough, etc., are constantly spread by *contagion*. Constantly the proof seems augmenting that this contagion is carried by germs.]

After these general observations, we shall consider those surgical cases that give rise to septic infection. First come the cases where there is decomposition on recent wounds; it usually appears within the first three days whether in such cases there will be intense, unusual, local, and general infection. If the local infection merely evince itself in moderate inflammation, which soon leads to circumscribed suppuration, if the general infection be followed by moderate fever, the affection would come under the head of traumatic fever. But if the local infection be very extensive, with phlegmonous inflammation and putrefaction, and the general condition assume a character soon to be described, we call the state septicæmia. In other cases the reabsorption of putrid matter takes place from a traumatic or idiopathic extensive gangrenous spot (as from gangrene due to disease of the arteries); this is more frequently the case in moist than in dry gangrene. In the same way the requirements for the reabsorption of putrid substances exist, if after delivery the placental surface of the uterus becomes gangrenous; some of the cases of puerperal fever are septicæmia.

It will be evident to you that the term septicæmia essentially depends on the etiology, just like the group of "typhous" diseases and that mild septic-traumatic fever has the same relation to septicæmia that typhus febricula has to typhus; in fact, the name "septi febricula" has been proposed. Still, as typhus in its different form is characterized by its symptomatology and pathological anatomy, th is also the case in septicæmia, although in it the pathologico-anatomic appearances are slight. Now, what characterizes the course of septicæmia? The nervous symptoms deserve the first mention: the patients are apathetic and sleepy, if not entirely comatose; rarely there is fearful excitement, and occasionally maniacal delirium; at the same time the subjective feelings are good; the patients do not suffer much. The tongue is dry, often as hard as wood, which renders the speech very peculiar; the patients are thirsty, but rarely drink, on account of their great apathy. Not always, but very frequently, there is profuse diarrhœa, more rarely vomiting. At first there may be great sweating,

FIG. 71.



Fever-curve in septicæmia after extirpation of an immense lipoma from between the muscles of the thigh. Death.

later the skin is dry and flabby. The urine is scanty, very concentrated, and occasionally albuminous. As the disease progresses, the patient passes his urine and fæces in bed. Bed-sores over the sacrum occur early. The fever (as shown by the bodily temperature) at first rises high; in acute pure septicæmia intercurrent chills *never* occur in the course of the disease, and initial chills are very rare. In the prognosis of septicæmia the conditions of the pulse and tongue are more important than the temperature. A small frequent pulse and dry tongue are bad signs; while a normal temperature has no prognostic value, very high or very low temperature makes the prognosis worse.

This is the usual course of acute pure septicæmia from recent injuries; but the patient may die in the first stages, with rising temperature. Cases also occur where the onset of the fever is scarcely marked by an elevation of temperature, and lastly some cases run their course without fever or with abnormally low temperature; the latter occurs especially in old persons with spontaneous gangrene; but the other symptoms above mentioned usually exist. From this and particularly from the above curve, we see that failing of the temperature of itself is by no means a sign of improvement, but that the other constitutional symptoms (strength, mental state, tongue, pulse, etc.) must also be taken into consideration.

I hope that, from what has been said, you have formed a true idea of septicæmia. Where the symptoms of the disease are marked, the prognosis is very bad; we shall speak of the treatment at the end of this section.

We now come to the *post-mortem appearances.* Occasionally it is difficult for us to recognize on the cadaver the œdematous infiltration and brownish discoloration of the skin that we observed about the wound during life. In other cases that had a long course (six to eight days) we find the subcutaneous tissue infiltrated with bloody, serous fluid; where the course is still longer (two weeks or more) the disease shows itself mostly by extensive suppuration of the cellular tissue, with more or less extensive gangrene of the skin. Frequently the internal organs present no morbid appearances. If there was continued profuse diarrhœa during life, you find swelling of the solitary and conglobate intestinal follicles. The spleen is often enlarged and softened, rarely it is of a normal size and firmness; the liver is usually full of blood, relaxed, and very friable, but without further change. In the heart the blood is lumpy, half-clotted, tarry, and rarely firmly coagulated, buffy; in most cases the lungs are normal. Sometimes we find diffuse single or double pleurisy of moderate extent, and also traces of pericarditis. Under pyæmia we shall speak more fully of these diffuse metastatic inflammations which are not due to emboli ere it is not very necessary to do so any more than it is to treat of mbolic infarctions and putrid abscesses, which are exceptionally found in septicæmia when the patients resist the disease a long time, and venous thrombi have occurred about the wound or gangrenous spot. As nothing special has been found on chemical analysis of the blood from the bodies of such cases, it must be acknowledged that what we find *post mortem* adds little that is characteristic to the picture of the disease, which is essentially etiologico-symptomatological; if we have not seen the patient during life, we shall often examine the dead body in vain for some palpable cause of death.

3. *Suppurative Fever, Pyæmia.*—Pyæmia (the name was formed by *Piorry* from πύον, pus, and αἷμα, blood) is a disease which we suppose to be due to the absorption of pus or its constituents into the blood; it holds the same relation to simple inflammatory and suppurative fever that septicæmia does to simple primary traumatic fever; it is symptomatologically characterized by intermittent attacks of fever, and in its pathological anatomy by the frequency of metastatic abscesses and metastatic diffuse inflammations. Other names for this disease are: metastatic suppurative dyscrasia, pus disease, purulent diathesis.

To give you at once an approximate picture of this disease, I will describe for you a case of pyæmia.

A wounded patient enters the hospital with a compound fracture of the leg just above the ankle. The injury has resulted from the fall of a heavy body. You examine the wound, find an oblique fracture of the tibia, but consider the injury of such a nature that it may heal. So you apply a dressing; at first the patient feels very well; he has but little fever till about the third or fourth day, then the wound becomes more inflamed, secretes relatively little pus, the surrounding skin becomes œdematous and red, the patient grows very feverish, especially toward evening, the swelling about the wound increases and slowly spreads, the whole leg grows swollen and red, the ankle-joint very painful; on pressure over the leg, a thin, bad-smelling pus flows slowly from the wound; the swelling remains limited to the leg; there is no trouble of the mind, no sign of intense, acute septicæmia; the patient is exceedingly sensitive to every dressing, he is restless and discouraged; there is febris continua remittens, with high evening temperature, and frequent, full, tense pulse; the appetite is lost, and the tongue heavily coated. This would be about the twelfth day after the injury. Quantities of pus flow from different parts of the wound; somewhat above it fluctuation is distinct; this collection of pus may be evacuated through the wound by careful pressure, but the escape is greatly impeded, and an incision must be made at the above point. This being done, a moderate quantity of pus is evacuated; a few hours later the patient has a severe chill, then dry burning heat, and, lastly, profuse sweating. The appearance of the wound improves somewhat; but this does not last long; we soon notice a new abscess near the wound, but rather behind it in the calf; there is another chill; more counter-openings are required at different spots to give exit to the pus, which forms in quantities. The left leg is the injured one; some morning the patient complains of great pain in the right knee-joint, which is somewhat swollen, and is painful on every motion. The nights are sleepless, the patient eats very little, drinks a great deal, and becomes much debilitated; he emaciates, especially in the face, the color of the skin changes to yellowish, the chills recur; the patient then begins to complain of pressure on the chest; he coughs some, but raises little sputum; on examining the chest, you find a moderate pleuritic exudation on one or both sides, from which, however, the patient does not suffer much, but he complains more of the right knee, which is now much swollen, and contains a great deal of fluid; as the patient sweats a great deal, the urine becomes very concentrated, and is occasionally albuminous. Finally, there is decubitus, but the patient does not complain much of this; he lies quietly, half insensible, muttering to himself. This would be about the twentieth day after the injury; the wound is dry, the patient looks miserable; the face, and especially the neck, is emaciated, the skin is very jaundiced, the eyes dull, the trembling tongue is perfectly dry, the skin cool, the temperature low, and only elevated at evening, the pulse small and frequent, the respirations slow, the breath of a peculiar cadaveric odor; the patient becomes entirely unconscious, and may, perhaps, remain so for twenty-four hours before death. On *autopsy*, you find nothing pathological in the skull; heart and pericardium normal; in the right auricle and ventricle a firmly-coagulated, white, fibrinous clot; both pleural cavities are filled with a cloudy, serous fluid; the surfaces of the lungs are covered with a net-like layer of jaundiced fibrine; on tearing this off, under it, in the substance of the lung, but particularly on its surface, you find quite firm nodules, as large as a bean or chestnut. These are found chiefly in the lower lobes; sections through them show that they are mostly abscesses. The parenchyma of the lungs, somewhat condensed, forms the capsule of a cavity, which is filled with pus and disintegrated lung-tissue; others of these nodules are bloody red, and, on section, the cut surface is somewhat granular, and in their midst there are occasional spots of pus of various size, and it is evident that they change to abscesses. They are the *red infarctions*, terminating in abscesses, with which you are already acquainted. Some of these abscesses lie so near the surface that they implicate the pleura, and the pleuritis is secondary. The liver is quite vascular and friable, but is otherwise apparently normal. The spleen is somewhat enlarged, and, on section, shows a few firm, wedge-shaped nodules, with their points inward, and their broad outer ends along the surface; they resemble the red infarctions of the lungs, and within they also have partly broken down into pus. The intestines, urinary and genital organs, show nothing abnormal. An incision into the right knee, which was painful during life, evacuates a quantity of flocculent pus; the synovial membrane is swollen, and in part hæmorrhagic, injected; the lustre of the articular cartilage is dulled. Examination of the wound shows little more than we found on the living patient; that is, extensive suppuration of the deep and subcutaneous cellular tissue, as well as pus in the ankle-joint; the walls of all these collections of pus consist mostly of broken-down tissue, true granulation has only occurred at a few points. The fracture is, however, more complicated than had been supposed, for a longitudinal fissure reaches to the ankle-joint, and on the posterior aspect of the tibia, which we could not examine during life, there are several detached fragments of bone. In the veins of the leg there are old plugs of fibrine here and there, also yellow puriform detritus, and in some places pure pus.

Let us make some reflections on this case, and suppose that you have seen a series of such cases, so that you are convinced that it is not an accidental association of various diseases, but a regular combination. You have an extensive, steadily-increasing suppuration in an extremity, with intense continued fever, which has exacerbations. To this are added suppuration in some distant joint, and circumscribed inflammations, ending in formation of abscesses in the lungs and other organs. These multiple points of inflammation keep up the fever, and they disturb the functions of the affected organs, and the patient dies of exhaustion. The peculiar and essential feature, as you will readily see, is the appearance of various points of inflammation, after the primary suppuration has attained a certain grade. You know the explanation of the occurrence of metastatic abscesses: they are always caused by venous thrombosis and embolism; it is unnecessary to recur to this. It is more difficult to explain the *diffuse metastatic inflammations* which occur both in septicæmia and pyæmia; they by no means always depend on abscesses of the lungs, as does pleurisy in the cases above mentioned; there are metastatic diffuse abscesses of the eye, cerebral membranes, subcutaneous tissue, joints, periosteum, liver, spleen, kidneys, pleura, pericardium, etc., which are independent of abscesses or emboli. The occurrence of these metastases cannot always be exactly explained. If the metastatic disease be nearly united to the original abscess, it might be attributed to conduction of the inflammation from the latter, possibly through the lymphatic vessels; as in cases where, after amputation of the breast or exarticulation of the humerus, there is pleurisy of the same side, or a fracture of the lower third of the leg is accompanied by suppuration of the knee-joint. In other cases it is possible that a part already diseased, or predisposed to inflammation, becomes acutely affected, as a result of the general febrile disturbance; for instance, sometimes fracture callus, say of the radius, that is already tolerably firm, suppurates in the third or fourth week, if the patient becomes pyemic from a complicated fracture of the leg, or from a bed-sore. But there are many cases where, as above stated, such explanations prove insufficient. Then we try to satisfy ourselves that there was a predisposition to inflammations, especially to suppuration in certain organs, which is necessarily accompanied by pus-poisoning; that the pus-poison circulating in the blood has a specific phlogogenous action on certain organs. I can give you no farther explanation on this point, but would like to render this hypothesis a little more plausible to you, by comparing it with analogous observations on the specific phlogogenous action of certain drugs, of which we have already spoken when treating of the etiology of inflammation, and its toxic-miasmatic causes, and their mode of action (Lec. XXI). *Diffuse* metastatic inflammations of internal organs are rare, unless among them we include the diffuse enlargement of the spleen, which is frequent, if not constant, in pyæmia. The diagnosis of metastatic abscesses and inflammations is easy, where they lie at the surface of the body and extremities; metastatic meningitis or choroiditis is relatively easy to recognize. The diagnosis of metastases to the lung may prove difficult; the foci are often so small and so scattered in the lung that they cannot be detected by percussion; the accidental pleuritic effusion often aids in the diagnosis of metastatic pulmonary abscesses; if there are bloody sputa and severe bronchial catarrh, the diagnosis may be considered certain; the subjective symptoms are often very slight; the dyspnœa is only severe when there is extensive pleuritic effusion. In pyæmia there is often more or less jaundice. It is not yet fully determined whether, in these cases, the coloring matter of the bile is formed from the red coloring matter of the blood without the intervention of the liver, or if icterus ever can occur without the liver having something to do with it, although most observers regard it as always being hepatogenous. At all events, icterus in pyæmia does not admit a diagnosis of abscess of the liver; this may be suspected if there be great pain in the hepatic region, but, instead of the expected hepatic abscess, I have, in such cases, occasionally found acute diffuse softening of the liver, which was accompanied by almost bronze-like icterus. Enlargement of the spleen may sometimes be diagnosed by percussion. Occasionally, albumen, with epithelial and gelatinous casts and blood in the urine, especially if there be considerable coincident decrease in the amount of urine excreted, justifies a diagnosis of acute metastatic nephritis; but during life it cannot be certainly determined whether the kidney has numerous metastatic abscesses or is diffusely inflamed, as may also occur metastatically. Pulmonary and splenic abscesses, as well as articular inflammations, are the most frequent, while those of the liver, kidneys, and other parts above mentioned, are far more rare.

There is one symptom of pyæmia that we must study more carefully, viz., *chills.* They occur irregularly, rarely at night, although they may come at any time of day, and their duration and intensity vary exceedingly; sometimes the patient only complains of slight chilliness and temporary shivering, sometimes he trembles and chatters his teeth as hard as in "chills and fever." At first the chills come rarely, then more frequently, two or three times daily; toward the end they again abate. The attacks themselves resemble those of intermittent fever in regard to chill, dry heat, and sweating; but after the attack there is no complete cessation of the fever, it almost always continues to some extent. Now, what is the true nature of this chill? When we have opportunity to make observations on ourselves we find that there is a spasmodic contraction in the skin; we must spasmodically knock the teeth together, even against our will; if this ceases for a moment, we do not feel cold, but rather hot, and the feeling of chilliness is more in the imagination, for otherwise we only have similar sensations and spasmodic trembling as an effect of great cold. During the chill the limbs and skin feel cold, as the blood has been driven from the capillaries by the spasm of the cutaneous muscles. But if you measure the bodily temperature with the thermometer from the commencement of the chill, you find that the temperature rises constantly and rapidly, occasionally from 3° to 5° Fahr., in a quarter or half an hour. At the end of the chill, and during the period of dry heat, the bodily temperature usually attains its highest point; it may reach 108° Fahr., but rarely goes over 104.5° Fahr.; from this point it gradually declines. The rapid increase of temperature is always in proportion to the phenomena of the chill; a certain irritability of the nervous system also appears necessary for its occurrence, for in torpid or narcotized persons chills are much more rare than in very irritable subjects (see page 184).

The most varied acute diseases begin with chills and fever, especially the acute exanthemata, pneumonia, lymphangitis, etc.; more rarely the acute miasmatic infectious diseases, such as typhus, plague, and cholera. Usually, however, these chills are not repeated, but only the onset of the disease is accompanied by this symptom; it seems as if the first entrance of certain pyrogenous substances into the blood of persons otherwise healthy was especially apt to induce chills, or as if certain infectious materials entering the blood excited particularly intense fever with chills. Hence, although we cannot consider chills a characteristic of pyæmia, still their frequent recurrence, as well as the generally *intermittent* type of the fever, is peculiar to this disease. Intermittent fever is the only disease in which we see any thing similar; there we have intermittent attacks of fever with regular intervals; we do not know on what this interval depends, but I should consider the immediate cause of the attacks of fever to be paroxysmal pouring out of morbid products from the spleen; in melanæmia and pigment metastases we have anatomical evidence that in intermittent fever substances pass from the spleen into the blood; it is known that collections of normal secretion occur in the pancreas and spleen, and are poured out during digestion; hence, it does not seem to me too bold to assume that, with these physiological evacuations of certain substances from the spleen, pathological products may also enter the blood. Thus, in pyæmia, from time to time pus or its constituents might be poured into the blood, and under otherwise favorable circumstances fever and chills might be induced. Extensive progressive inflammation about the wound must be regarded as the chief source of such repeated purulent infection; destruction of the granulating surface by frequent injury, rapid destruction of the granulations by chemical agents, any new progressive inflammations occurring about the wound, may open an entrance for the pus into the lymphatic vessels which have been closed; new inflammation may cause suppuration of the coagula in the lymphatic vessels, and the pus from these may enter the blood; it might also be imagined, although difficult to prove, that in venous thrombosis the central coagula enclosing the pus in the veins are torn loose, and the pus is swept into the blood through a passable collateral vein, which opens farther on; this might be caused by muscular contractions. Lastly, metastatic inflammations, whether due to emboli or not, also induce new attacks of fever; but that this is not the only cause is proved by occasional autopsies on cases that have died from intermittent purulent fever, after ten or twelve chills, where no metastatic inflammations have been found; the cause of the repeated chills may then lie in the mode of extension of the local process, or be hidden in the bones or elsewhere. Statistics greatly favor the idea that the chills depend on new inflammations, for they show that the chills (or at least the intermittent fever attacks, which may occur without chills) occur far more frequently in persons in whom subsequent autopsy shows inflammation of internal organs than in those where this is not the case. It must be mentioned, as a matter of observation, that chills occur almost exclusively in the commencement of acute inflammations, and are intermittent only in intermittent fever and reabsorption of pus, while they do not occur in acute septicæmia. Probably the chemical qualities of the infecting matter here play an important but unknown *rôle*. Unfortunately, experiment here leaves us entirely in the dark; I have never succeeded in exciting chills or intermittent attacks in rabbits, dogs, or horses, by injections of putrid substances or good pus; pus and putrid matter have the same action on animals, as regards fever; we can only artificially excite the intermittent course of the fever in animals by repeating the injections.

From what you have just heard, you will understand that the usual method of measuring temperature morning and evening can give no picture of the course of the fever in pyæmia; for in this way the measurement may fall at one time in the acme, again in the defervescence of an attack of fever, or at another time in the remission (complete intermission of the fever rarely happens in pyæmia); thus we would of course have very irregular fever-curves. To obtain an accurate picture of pyæmic fever, it would be necessary to leave the thermometer constantly in position, and to note the temperature every hour or so; as this would greatly annoy the patient, and we have enough other signs to decide the prognosis and treatment, I have been unable to make up my mind to do this. The investigations as to whether pyemic pus contains peculiar substances, or its qualitative composition differs from that of the pus in persons who recover without any complications, have thus far proved without result. The pus of pyæmic patients does not always smell bad, nor always contain cocci; still cases where putrid pus containing cocci enters the circulation are the more frequent. We do not know whether the pus coccus grows after entering the blood. I have not found cocci and bacteria in the blood of pyæmic patients.

The mode of onset of pyæmia varies in some respects. Most frequently this disease, which we regard as a peculiar, malignant form of suppurative fever, begins when suppuration begins, or later, when new inflammations occur about the wound, whether they be immediately connected with the traumatic inflammation, or occur accidentally after the point of traumatic inflammation has been bounded. Then the pyæmic fever develops from the traumatic fever, or from the secondary fever, and in such cases these are considered by some observers as prodromal stages of pyæmia. The moment when the paient becomes pyemic cannot be decided any more accurately than can the passage of primary traumatic fever into septicæmia. I retain the designation "pyæmia" for the disease just described. I have told you that the reabsorption of pus is the cause; intermittent course of the fever, with rapidly-increasing marasmus, the chief symptom; and the metastatic inflammations very essential anatomical conditions; but it is sometimes very difficult to decide whether a given case shall be termed severe traumatic fever, septicæmia; or severe suppurative fever, pyæmia. The chills may not occur; then it is difficult to determine the intermittent course of the fever; the metastases may not be diagnosticated during life. If you have a case of osteomyelitis with frequent chills, if the patient dies and you find no metastases, is that pyæmia? Or an old marasmic man has a compound fracture; he dies with symptoms of complete exhaustion in the fourth week, without having had very high fever or chills; you find no metastases; is that pyæmia? For the beginner who would like to have every thing well systematized, these questions, and their doubtful answers, are very embarrassing. You will find surgeons who call the above cases pyæmia, others who term them simply intense suppurative fever or febrile marasmus. If you adhere to the above description, and have correctly comprehended the relation of infection to venous thrombosis and embolism, it is to be hoped you will not be perplexed about the names. Indeed, it is scarcely possible to make a name for every link between septicæmia, purulent infection, diffuse metastatic inflammations, thrombosis, embolism, etc. For instance, septicæmia occurs without a trace of metastases, with diffuse metastases, with thrombosis and embolism; purulent infection without a trace of metastases, with diffuse metastases and thrombi, with thrombi alone, with thrombi and emboli; there are thrombi with local sequences without emboli, with emboli, with hæmorrhagic effusions, with apoplexies, etc. Besides the words already given, some others have been introduced to designate combinations of the various processes. For pure purulent infection (infection with thin, bad pus—ichor) *Virchow* has proposed the name *ichorrhæmia*. *O. Weber* uses the name *embolhæmia* for the condition in which emboli are found in the blood. The classification given by *Heuter*, in his excellent work on this subject, appears to me very practical. In pure cases of purulent infection without metastases he calls the disease "pyohæmia simplex;" in cases with metastases, "pyohæmia multiplex."

The *course* of purulent infection is usually acute (8–10 days), often subacute (2–4 weeks), rarely chronic (1–3–5 months). The rapidity of the acute cases is due partly to the intensity and frequent repetition of the infection, partly to the extent of the metastases

The chronic cases usually occur in very strong or tough patients, and the infection is only moderately intense, and not often repeated; the metastases are in external parts, as abscesses in the cellular tissue, and suppurations of the joints, which keep the patient sick after the other results of purulent infection have disappeared. The *prognosis* essentially depends on the course. The more frequently the chills are repeated, the more rapidly strength is lost; the earlier the symptoms of internal metastases present themselves, the sooner the patient will die. The longer the intermissions between the exacerbations of fever, the better the strength is preserved; the longer the tongue remains moist, the more hope we have of the patient's recovery; he is not out of immediate danger till the wound again looks well, till he has been entirely free from fever for several days, and has otherwise the appearance of a convalescent. It is exceedingly rare for a patient who presents *all* the above symptoms of decided pyæmia to recover.

[It is only in the last ten years that accurate diagnosis between septicæmia and pyæmia has been possible, and this is based on the etiology, clinical course, and anatomy; now this differentiation is assailed. It is asserted that the poison is the same in traumatic fever, septicæmia, and pyæmia; that it is always the product of coccus proliferation; but I cannot entirely agree with this view. Quite recently we have had an excellent work on this point by *Robert Koch:* he induced traumatic infectious diseases in animals, and then examined their tissues microscopically. He thinks the different infectious processes, septicæmia, progressive gangrene, pyæmia, and erysipelas, are due to different forms of vegetation which may be recognized as distinct forms of cocci by the microscope. *Koch* thinks that, besides the fungi acting deleteriously and multiplying in the organism, there are others which are either not injurious for certain animals, or else do not develop in animals.

Although *Koch* originally inoculated with putrid fluids, he comes to the conclusion that it is possible to transfer a certain species or various forms together to the same animal, and thus induce one of the above diseases or a combination of several of them. Satisfactory as it might be to do so, it is not yet possible to refer the traumatic infectious diseases in man to carefully-studied, characterized contagions; although there is great probability of the existence of different infecting materials, and of the parasitic nature of at least part of the accidental diseases of wounds. But the clinical picture of the above states is in most cases varied enough to distinguish them; and should it once be decided that the difference was only due to more or less intense action of the same chemical process, it would be very satisfactory scientifically, though it would not diminish the clinical and prognostic value of the descriptions of the diseases. From individual observation I can confirm *Hueter's* proposed name of "septo-pyæmia" for a disease where the clinical symptoms of septicæmia and pyæmia pass into each other. The term "subacute pyæmia," used by *Stromeyer* and others, corresponds to septicæmia. The French "gangrène traumatique foudroyante" is accompanied by development of gas deep in the muscles, green discoloration, and rapid decomposition of the tissues during life; it is very rare; I have only seen two cases after amputation of the thigh for severe injuries. In some such cases the gangrene is caused by the injury, as when the tissue has been extensively crushed and killed, without the fact being recognized at the time of the injury. But in other cases contusion might be excluded and still the gangrene, with development of gas, proceeded rapidly. Probably, under especially favorable conditions, there may be developed substances of such activity as snake-poison, etc., whose contact at once destroys this life of the cells.

We can rarely recognize purulent breaking up of the thrombosis in a vein; if a few days after an amputation the patient has a chill, we may suspect a purulent infiltration of a venous thrombus. In some such cases, exposure of the chief venous trunk, pinching it together and dividing its wall, turning out the coagulum and cutting out the diseased part of the vein, applying a ligature in the healthy tissue, has arrested the general infection and saved the patient. *Hunter* suggested compressing the veins in suppurative thrombophlebitis, but unfortunately this is only practicable in a few cases, as after amputation; while it is scarcely possible, in pyæmia after complicated fractures, to recognize early the location of the decomposing thrombus, unless it be in some large subcutaneous vein. In acute sepsis the local treatment is most important. Bad as are the results of amputation in such cases, we still have to operate and risk seeing the stump become gangrenous. In gangrène foudroyante, or gangræna septica acutissima, energetic antiseptic treatment, multiple incisions, removal of gangrenous tissue, applications of a five-per-cent. solution of chloride of zinc, and permanent irrigation, may save the patient. At all events, do not be deterred by the apparent hopelessness of the case from trying something.]

The severest cases of septicæmia are those where toward the middle or end of the second day great cyanosis and collapse come on rapidly; then death usually occurs in a few hours. Such patients look like those in the algid stage of cholera, only in septicæmia vomiting and persistent diarrhœa are rare; after getting along well perhaps for twenty-four hours after the operation, the patients seem as if poisoned. In these cases (which may be accompanied by diphtheria) the secretion from the wound does not always smell disagreeably. It cannot be shown that the intoxicating matter in these cases is different from usual, or that the inflammatory alteration of tissues causes a remarkable amount of poisonous product. According to the above, the variety of symptoms in septicæmia is considerable; but this proves nothing against the claim that the septic poison is always the same, for there is the same difference from cholera, carbuncle, diphtheria, and bites of serpents, in which cases we do not assume different natures, but only differences of intensity and in the quantity of poison absorbed, and difference of resisting power in the patients.

Some surgeons prefer to say that a patient who has been wounded or operated on has died of severe typhous traumatic fever, instead of using the term "septhæmia" or "septichæmia." This is an incorrect expression even if it were practically true. "Typhous" is used in the old sense, like the τῦφος of *Hippocrates*, for stupid; later the term typhous was applied to fevers in which the patient was stupid; during the last twenty years well-characterized infectious diseases have been called "typhus." It is better to use the name thus, and not bring the term typhous into use again. *Virchow* uses "ichorrhæmia" in the same sense that I do septicæmia; ἰχώρ means blood-water, lymph, serum of wounds; the older surgeons occasionally apply the term to thin, bad pus.

[Since antiseptic treatment of wounds is in vogue, septicæmia and pyæmia are fortunately rare; while the former were common even in the best arranged hospitals, now where antisepsis is common months may pass without a case, and even this is apt to occur in patients infected before entering the hospital. The whole difference between formerly and now lies in the management of the wound and paying attention to trifles.

When the disease is fully developed little can be done for it.]

The cause of this is said to be that the septic matter, once taken into the blood, acts as a ferment, and that a small quantity suffices to cause decomposition of the blood and all the juices. As already stated, I do not consider this hæmatozymotic action of septic poison as proved; on the contrary, I think that it, like the poison of diphtheria, malignant pustule, etc., often acts for so long a time and so differently, even when taken up in small quantities, because the human organism (as well as that of some animals) only sets it free very slowly, and because at the points where it is retained in the body it often excites new foci, where the poison forms anew (perhaps less intense). For instance, I think dogs can bear so much septic poison because they pass it off so rapidly by the bowels; they thus escape even very severe putrid infection. The power of getting rid of absorbed infecting poison more or less rapidly may vary with the individual to some extent. The same view would hold in typhus, cholera, and the acute exanthemata.

[Many surgeons assert that pyæmia often results from a miasm, especially from that developed by many wounded patients lying together; this view is based chiefly on the fact that where numerous severe surgical cases lie together, many of them die of pyæmia, and that even mild cases with granulating wounds become pyæmic under such circumstances. I can entirely agree to the miasmatic origin of pyæmia, if by miasma is understood what I understand by it in the present and some other cases, namely, dust-like dried constituents of pus, and possibly also accompanying minute living very small organisms, which in badly-ventilated sick-rooms are suspended in the air or adhere to the walls, bedclothes, dressings, or carelessly-cleaned instruments. These bodies, which are in some respects of different natures, are usually phlogogenous, all pyrogenous when they enter the blood; of course they will collect chiefly where there is the best opportunity for their development and attachment, that is, in badly-ventilated sick-rooms, where the patients are carelessly attended and remain some time in the same apartments which are not kept sufficiently clean.]

Another common question is, *Is pyæmia contagious?* According to the view I have just given of pyæmic miasm, this is answered to some extent both in the affirmative and negative. A fixed molecular miasm, originating from a suppurating pyæmic patient, must at the same time be regarded as a fixed contagion; but according to my view this miasm may just as well come from a non-pyæmic patient; then it cannot be termed contagious in a specific sense, for a contagion always induces the same disease. You see that the strife as to the contagiousness or non-contagiousness of pyæmia must go back to the views as to the nature of the disease; it is only important for those surgeons who regard pyæmia as a peculiar specific disease, not related to suppurative fever—a view which I regard as groundless and practically useless, and against which I have long fought, and I hope with some success. With all these things arises the question, *Does pyæmic miasm enter the body only through the wound, or also through the skin and mucous membranes?* Although the latter is not impossible, I have not yet made any certain observations by which such an hypothesis can be considered proved or even probable; but from my experience I hold to the opinion that the infection of the whole body comes from the wound, whether poison finds circumstances favorable to its development in the wound and surrounding parts, or whether it be introduced into the wound already developed. I am not shaken in this view even by those [illegible] cases where there is no visible change, or only very little, in the wound on commencing pyæmia, for possibly the infecting body has very little, if any phlogogenous action, and hence may enter the blood through the wound, and have a pyrogenous action, without causing any change in the wound at its entrance. *Sex* seems to have very little influence on the frequency of infectious diseases of this class; possibly temperament, the energy and frequency of the contractions of the heart and arteries, may have more influence on the reabsorption of the deleterious substances. Judging from general impressions, children seem less disposed to pyæmia than adults. It would be exceedingly difficult to make statistics on this point, as so few severe injuries occur in women and children as compared with men; consequently, the fact that so many more men die of traumatic-infection fever of course proves nothing about the predisposition of either class to this disease. *Open* wounds of bone particularly dispose to pyæmia; judging from my experience, those wounded in the lower extremity are most, those wounded in the trunk are least, in danger of becoming pyæmic. The time of year and the collection of severely wounded in hospitals seem to have little if any direct influence on the development of pyæmia, unless by causing greater accumulation of infecting matter in the dressings, etc., thus increasing opportunity for infection.

Lastly, I must mention the so-called spontaneous pyæmia. Cases occur where multiple abscesses (of the subcutaneous tissue, for instance), or even venous thrombi with embolic metastatic abscesses, appear without our being able certainly to detect any primary point of suppuration; these cases, especially if they run an acute course, are called spontaneous pyæmia. There is no reason for raising a new theory for these rare cases, where we simply fail to detect the primary point of inflammation; I doubt not that there will hereafter be less mention of these cases, which, according to old theories, were very enigmatical, as we are constantly learning to observe more accurately, and, on more careful examination, shall usually find the connection of the symptoms.

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From the intimate relation, which we suppose to exist, between traumatic fever, septicæmia, and pyæmia, it seems correct to speak of the *treatment* of these diseases under the same head. This may be divided into prophylaxis, and the treatment of the developed disease. The former is by far the most important; it consists in avoid ing every thing that may favor the disease. Even in operations there are some points to be observed; all the instruments used, the hands of the operator and his assistants, and the sponges (which should either be perfectly new or should be replaced by moist compresses), should be perfectly clean; hæmorrhages should be entirely arrested, especially if sutures are to be applied, and the wound is deep; if the wound heals by suppuration, the compresses should be moistened with chlorine-water. In accidental injuries, all deep wounds, particularly if contused, should be kept quiet by dressings; all that is necessary in compound fractures has already been said. Every thing that can excite secondary inflammation (page 178) should be most carefully avoided; the patient should lie quiet, and as comfortably as possible. I would remind you of the treatment previously given for contused wounds. Of course the greatest care must be used in dressing the wound; here the greatest pedantry may be very beneficial. Hospital influences, which I only touch on here, are peculiarly interesting. Although few of you may have the fortune to control civil hospitals, any of you may desire knowledge on this point during war. Of course, hospitals should only be located where there is no marsh miasm. The hospital should be placed in a large, open space, with trees planted about it, and should have properly-located odorless water-closets. Of all artificial systems of ventilation, I think that *Van Heke's* is the only one worth any thing. In it the walls of the whole building are traversed by canals, opening into every ward. All these canals start from cross-passages under the building, at whose points of intersection there is a sort of wind-mill, driven by steam, so that new air is thus constantly driven into the wards of the hospital (pulsionssystem). If there be no artificial system of ventilation, we must do as well as we can with the so-called natural ventilation, i. e., corresponding draught-openings should be made above and below in doors and windows, so that in their beds the patients may escape the draught as much as possible; these ventilators should never be entirely closed. An excellent English surgeon, *Spencer Wells*, says: "There is only one true means of ventilation: the impossibility of closing doors and windows." I consider a proper use of the wards is important as their ventilation. No surgical ward should be used more than four weeks in succession; it should then be emptied for a ew days and carefully cleaned; the walls should be painted with oil-aint so that they may be washed, or else they should be white-washed at least two or three times a year, more frequently if neces-ary. The beds should be frequently aired, shaken up, and sunned, and the straw in the sacks often renewed. Every surgical division should have one, or, still better, two supernumerary wards, so that they may be regularly occupied in turns. With the same obj there should not be more than six or eight beds in one ward, so t enough patients may be discharged every week to empty one ro The new patients should always be brought into the ward l cleaned. This is the only way to prevent the extensive developm of miasm in hospital. To attain the best possible results in hosp we must have plenty of room, and plenty of money for nurses, li etc. We can thus use even badly-located hospitals. Large wa with twenty or thirty beds, which, from press of patients and o causes, cannot be emptied at will, are very unsuitable. The director of a surgical division should, above all things, have at his disposal a large number of well-ventilated rooms of medium size, which can be emptied and cleaned at certain times. Bad hospitals, and especially badly-kept rooms for surgical patients, are worse than the poorest tenements; they may become slaughter-pens for the wounded. Surgeons should never forget that they themselves are often to blame if their patients have erysipelas, hospital gangrene, diphtheria, etc.; for, if, after old customs, we ascribed every thing to the invisible, omnipresent, intangible, ethereal miasm and genus epidemicus, it would be death to all our future progress.

Coming now to the treatment of traumatic fever, septicæmia, and pyæmia, we may say that, for simple traumatic and suppurative fever, which does not pass the usual limits, we generally use nothing but cooling drinks, fever diet, and a little morphine at night to secure good rest. If the fever lasts longer, or assumes a peculiar character, we may resort to febrifuges. Digitalis is here of little use, on account of its slow, uncertain action. Veratria reduces the temperature, but appears to do little good in toxic traumatic fevers; still, further observations must be made on this point, especially in pyæmia. The accurate studies of *Biermer* show that this remedy should be used very carefully. Formerly aconite was highly recommended in pyæmia by *Textor*. I have seen no good from it. Quinine is the most efficacious remedy for the intermittent suppurative fever, especially in combination with opium; 6–8–16 grains of quinine in the course of the afternoon, and one grain of opium at night, often arrest the chills; in severe suppurative fevers I employ these remedies with benefit; in decided pyæmia they do less good. After careful observation, *Liebermeister* found that quinine only showed its antifebrile action in typhus and other infectious diseases with certainty when given to the extent of fifteen grains or more daily. There are plenty of observations, too, on remedies for directly opposing the blood-poisoning. I have found no effect from the antiseptic internal remedies, the acids, chlorine-water, and sulphurets of the alkalies (which are greatly praised by *Polli*).

But we may also use other remedies, intended, by increasing the change of tissue, to separate the organic poison from the blood. Seeing the profuse diarrhœa in dogs artificially made septicæmic, and finding them to recover frequently after these diarrhœas, we might suppose the poison to be most naturally excreted through the intestinal canal. In fact, *Breslau* has had favorable results from repeated doses of laxatives in puerperal fever. I am sorry not to have had similar experience in pyæmia. In this disease diarrhœa is a severe complication, which quickly induces collapse. It might also be thought advisable to increase the secretory activity by giving emetics; but they are followed by such collapse that we must be careful in their administration. In septicæmia I have often tried to induce profuse perspiration, when the skin was very dry. This was occasionally done by a warm bath, lasting for an hour, and then wrapping in blankets. This occasionally does good; indeed, I think patients have thus been saved that I had thought incurable. Further trials should be made with this remedy. Copious diuresis also may be induced by plenty of drink, but it has not much effect on the general condition. Lastly, we might think of arresting the further absorption of injurious substances from the injured or inflamed part by amputation, even after the appearance of severe constitutional symptoms. In acute cases of septicæmia and pyæmia this very rarely has a permanently beneficial effect, although there is almost always temporary improvement. But in subacute and chronic pyæmia amputation may, indeed, save life; unfortunately, however, such cases are rare.

So we finally come back to what we said at first, that much may be done to prevent severe traumatic and suppurative fever, but that there is little to be hoped from treatment of these diseases when fully developed.

## LECTURE XXVII.

**4. Tetanus; 5. Delirium Potatorum Traumaticum; 6. Delirium Nervosum and Mania.—Appendix to Chapter XIII.—Poisoned Wounds; Insect-bites, Snake-bites; Infection from dissecting Wounds.—Glanders.—Carbuncle.—Hydrophobia.**

THE group of diseases which belong to the traumatic and phlogistic infectious conditions, and of which we still have to speak, comprises tetanus, drunkard's madness, and the psychical disturbances which so rarely occur after injuries and operations. The views, as to their origin, vary greatly; as, from their symptoms, the processes in question would be referred to irritation of the brain and spinal cord, their cause is usually sought in the nervous centres. But it is known that by blood-poisoning, with strychnine, severe spasms, and with alcohol, psychical disturbances (drunkenness) may be induced; hence, it is very possible that the following forms of disease may result from poisoning with peculiar substances, which possibly are very rarely formed in wounds, and thence absorbed, while in drunkard's mania a series of ordinary pyrogenous materials may excite certain disturbances (namely, fever with peculiar, predominant psychical disturbances) in the organism already poisoned by alcohol. The symptoms that we shall see in these diseases are all present in ordinary fever, although to a slighter and less prominent degree; in the combination of the affected muscles, chills have an undoubted similarity to tetanus, psychical disturbances, even to maniacal attacks, occur as so-called fever delirium in some cases of septicæmia, but especially in typhus. In describing the individual diseases, we shall occasionally recur to these remarks, for which, unfortunately, we have no experimental foundation.

4. *Traumatic Tetanus* (*Trismus*).—This disease, which consists in spasms of the muscles of the jaw alone (trismus), or of all the muscles of the body (tetanus), the muscles of the extremities being most affected sometimes, at others those of the front or back of the trunk, occasionally occurs in the wounded; though it is rare in proportion to the traumatic diseases above described, it occurs still more rarely in persons without wounds. In large hospitals, years may pass without a case of tetanus being seen; again, at certain times, numbers of cases will appear, so that there has been an inclination to seek an epidemic cause. The disease is by no means confined to hospitals, but comes either in or out of them. However, before discussing the etiology, I will try to give you a brief description of an acute case.

The third or fourth day after an injury, rarely sooner, often later, you find that the patient cannot open his mouth well when speaking, and complains of tearing, drawing pains, and of stiffness in the masticatory muscles. In very acute cases there is high fever even with these first symptoms, in other cases the patient is free from fever at this stage. The lines in the patient's face gradually assume a peculiar, stiff expression, the facial muscles being to some extent spasmodically contracted. Subsequently there are tetanic spasms, which may affect the trunk or extremities; in some cases these last several seconds or minutes, and are induced by any external irritation, just as in hydrophobia. These spasms are accompanied by severe pain. Occasionally, from first to last, some groups of muscles remain regularly but painlessly contracted; in some patients the twitchings (shocks of *Rose*) are entirely absent, and there is only permanent contraction of more or less distinct groups of muscles. Not unfrequently the patient's body is bathed in sweat, his mind being clear; occasionally the urine contains albumen; sometimes the fever rises to a height that is rarely seen, even to 104° Fahr., or over. But I have seen cases of trismus prove rapidly fatal, without the temperature becoming elevated; *Rose* has made similar observations. Death may occur within twenty-four hours from the commencement of the disease, but the latter may also last with considerable severity for three or four days; these cases also are to be classed among the acute. There is a more subacute or chronic form of trismus, and of trismus and tetanus, in which there is merely a gradual development of a moderate trismus and of contractions without pain, extending to single groups of muscles of the injured limb. In these chronic cases fever is usually entirely absent. It is rare for an acute case to become chronic.

All the symptoms indicate that there is an irritation of the spinal medulla and of the portio minor of the fifth pair. The symptoms resemble, although remotely, those which may be induced by poisoning by strychnia. Unfortunately, the results given by autopsy of these patients are usually very unsatisfactory; in the acute cases, especially, nothing can be found in the spinal medulla; in cases of some days' duration, *Rokitansky* claims to have seen a development of young connective tissue in the spinal medulla, which would make it appear that there was an inflammatory affection of this nerve-centre. My examinations of the spine and nerves in tetanus have thus far given only negative results. In preparations made from cross-sections of the spinal medulla, and sent to me by excellent specialists in examining the nervous system (Dr. *Goll*, in Zürich, and Dr. *Meynert*, in Vienna), I saw the connective tissue remarkably developed at some places, it is true; but, as there was no collection of young cells, I was in doubt whether this increase of connective tissue was really new formation, or was due to mere accidental swelling. The symptoms during life, in cases where we find decided evidences of spinal inflammation, are so different from tetanus as to render it improbable that the latter depends on myelitis spinalis. The discovery of small extravasations of blood in the muscles and nerve-sheaths, on autopsy, shows little about the nature of the disease, for they may be caused by ruptures of the capillaries during the great muscular contractions.

There are many views as to the causes of this disease, as there usually are about affections with no anatomical, pathological characteristics. At first, it was natural to examine the nerves, and in many cases the nerve-trunks are crushed by the injury, or torn or irritated by foreign bodies. I myself have seen some such cases; a few years since, I saw a sporadic case where, in an open splintered fracture of the lower end of the radius, the median nerve was half torn through; the third day trismus ana tetanus appeared suddenly, and proved fatal i eighteen hours. It is no use to build theories as to how this partic lar variety of injury of the nerves should induce tetanic spasms, whi they are very rare after simple division of the nerves, for there a many cases where tetanus has arisen from simple wounds of the sk from granulating surfaces fully developed and cicatrizing, or ev after a blister, the sting of a bee, etc. It is, however, remarkable th the disease is particularly frequent after injuries of the extremit especially of the hands and feet, while it is rare after considera injuries higher up the limb and on the body. I also think that I have found the cases, where tetanus developed from granulating wounds, to be more chronic and milder than those where it has developed soon after the injury. *Rose* thinks that tetanus appears particularly in cases that are treated badly or not at all; my experience is opposed to this. After applying in vain to the nerves and tendinous tissue, the various changes of temperature were resorted to to explain the occurrence of tetanus; some said that it was favored by hot, sultry weather. I cannot altogether deny this view, for hitherto I have only seen numerous cases of traumatic tetanus in hot, sultry weather, but small epidemics of it have been seen in winter. Others ascribe the chief blame to catching cold from draughts or to rapid changes of temperature. Finally, there are still others who do not believe that the nervous system is primarily affected, but think that the blood first becomes diseased and acts secondarily on the nervous system. Within a short time *Rose* has resurrected an old idea, that tetanus, like hydrophobia, is to be regarded as a primary blood-disease. It cannot be denied that the two diseases are much alike; a proof of their being actually analogous would be most strikingly given by inducing hydrophobia, by inoculating animals with the blood or secretions from a tetanus patient. Of course, we should not think of inoculating another man. At present, I strongly incline to the humoral view of tetanus as due to a peculiar poison, although I have no proofs of it. At all events, the blood of a tetanus patient should be injected into a dog, to show whether tetanus may be transferred through human blood to a dog, and also whether it has a pyrogenous action; should tetanus appear in the dog, it might be regarded as proved that tetanus was a humoral disease; if the experiment be negative, it proves nothing against the humoral causes of tetanus, it only shows that the blood of a *man* with tetanus will not induce tetanus in a *dog;* it would still have to be decided whether the blood of a *dog* with tetanus, transferred to another *dog*, would prove as inactive. The fact that tetanus may be confined to one limb, or even to one hand as I have seen it, speaks in favor of a local cause, which may be limited to the nerves;

but there are also a localized lymphangitis, localized erysipelas, etc.; the fact that, after amputation, for instance, twitching not unfrequently occurs in the stump before the spasms become general, might also indicate that the tetanus-poison formed in the wound first irritated the muscles and nerves of the stump, and then passed to the spinal medulla. There still remains much to be investigated on this point. The high fever in most cases of acute tetanus, and the fact that the temperature rises even after their death, has greatly occupied pathologists; this became still more interesting when *Leyden* showed that great elevation of the temperature of the blood was caused in a dog in which tetanus had been artificially induced by passing a strong current of electricity through the whole spinal medulla. *A. Fick* showed that a surplus of heat was formed in the muscles, and thence distributed to the blood; also that the elevation of temperature, noticed in the rectum after death, was due to the equalization of warmth between the muscles and the rest of the body. If these experiments, which I have repeated, prove that tetanic muscular contractions considerably elevate the bodily temperature, they do not show that in traumatic tetanus in man the high temperature is solely or chiefly due to the muscular contractions; this view is opposed by the fact that very acute cases of tetanus may run their course almost without fever, although this rarely happens; here, too, there are many enigmas to solve.

Unfortunately, in most cases the *prognosis* is bad; very few of the acute cases recover; of the chronic cases, which last over a fortnight, some get well. Unfortunately, the latter are proportionately rare.

From the lack of knowledge about the etiology of this disease, the *treatment* can be only symptomatic. Numerous remedies have been recommended at various times. Generally, the treatment most resorted to is by narcotics, with opium and chloroform; this is the plan I have adopted. Opium is given in large doses, as high as fifteen grains or more in a day, or a corresponding quantity of morphine may be given, best by subcutaneous injection; sometimes this arrests the spasms, sometimes it does no good. At all events, the sufferings of the patient are lessened. During the attacks the patient may be greatly relieved by inhaling chloroform to narcotism. Under this treatment many cases have recovered. The general aim of the treatment is to alleviate the acute course, and make it more chronic, as this gives more hope of recovery. Among other modes of treatment, I may mention the frequent employment of warm potash-baths; and the application of strong irritants along the spine, large blisters, moxæ, the hot-iron, remedies from which I cannot promise any good effects, and, lastly, the curare, which is of late occasionally used, has not answered the hopes that some had of it.

In the chronic cases you need not employ any special treatment; the patient remains in bed, and should keep perfectly quiet; he should be guarded against all injurious influences, especially from physical or mental excitement.

5. *Drunkard's madness. Delirium potatorum traumaticum. Delirium tremens.*—We now come to an enemy of the wounded which, fortunately, is not very dangerous. You have doubtless heard of delirium tremens, the acute outbreak of chronic alcoholic poisoning, which may come on spontaneously, or from some acute diseases, especially pneumonia. Injuries are a frequent cause. You will become better acquainted with this disease from the lectures on medicine; as the attacks, from whatever cause they arise, are much alike, I shall be very brief on this point.

The disease generally breaks out within two days after the injury, in some rare cases it is longer. It only attacks patients who have for years been accustomed to the free use of alcohol, especially of schnaps and rum; but it is an error to consider beer and wine drinkers exempt from delirium. The first symptoms are sleeplessness, great restlessness, trembling hands, unsteady look, tossing about in bed, and talkativeness, and then delirium. The patients talk constantly, see small animals, midges, flies, etc., swarming about them; mice, rats, martins, foxes, etc., crawl from under their beds; they think they are in a smoky atmosphere, and feel dizzy. The delirium often has the most comical form; a soldier, whom I treated in Zürich for delirium tremens, saw numbers of other soldiers in his water-glass; when I entered the room, he spoke lowly to my assistant, taking me for his major, etc. Generally the hallucinations are of a happy nature, nevertheless, the patients are tormented with restlessness, constantly toss about in bed, and wish to get up. If we have not two stout nurses to hold these patients, there is often no way of avoiding the application of a strait-jacket and tying them in bed. These patients are usually good-natured in their delirium, and if spoken to emphatically they give sensible answers, but soon fall back into their wanderings. Of all kinds of injuries, fractures, especially open fractures, most frequently give rise to the outbreak of the disease, and, before we had firm dressings for such patients, it was a difficult task to fix the broken limb, as the patients did not notice the pain, and moved the limb so forcibly that any splints were loosened in a few hours. Even where there is marked delirium, the prognosis is not unfavorable, according to most surgeons; from my somewhat meagre observations, I cannot agree in this opinion: of the patients with acute delirium tremens that

I have treated, at least the half have died; they often declined suddenly, became unconscious, and soon died. Others recovered, especially when it was possible to make them sleep a while; this is the object of the treatment; opium in large doses is the almost universal remedy, for it we may substitute small doses of tartar-emetic. After this the patients fall into a comatose state, from which in favorable cases they awake cured, but sometimes sleep on till death. I can recommend no better remedy than opium in delirium tremens, although I must acknowledge that in large doses (gr. ii.—vi. every two hours till sleep is induced), I do not consider it free from danger [of late, hydrate of chloral, in doses of gr. xx.—ʒ i, is said to have been given with great benefit in such cases; it is claimed that it acts well not only on the delirium tremens, but on the fever which so often accompanies the injury]. Of late, there has been a great outcry in England against the opium and tartar-emetic treatment, and a more expectant treatment has been recommended. Others have had good results from digitalis; most surgeons are well satisfied with the opium-treatment, and the coincident administration of strong wine and cognac has been highly recommended. The more chronic cases of delirium potatorum, without maniacal attacks, have seemed to me of more favorable prognosis; there, strong grog is useful; I give the following mixture: one yolk of egg, one ounce of arrack, four ounces of water, two ounces of sugar; this does not taste badly, and may also be used as a stimulant for old persons (a tablespoonful every two hours). I must warn you against abstracting blood, which is very dangerous in drunkards, and not unfrequently induces collapse terminating in death.

Autopsy of patients who have died of delirium tremens shows no special cause of death; we find the changes common to topers; chronic gastric catarrh, fatty liver, Bright's kidneys, thickening of the meninges of the brain, but no constant changes in the brain-substance proper.

6. *Delirium nervosum and psychical disturbances after injury.*—By *delirium nervosum traumaticum* we mean a state of excessive nervous exaltation without fever, occurring after injury; this is said particularly to affect hysterical persons. I have only seen one case to which I could apply this name: a man twenty-four years old (from Canton Thurgau, the land of perry), who had never been accustomed to drinking, after a fracture of the leg, complicated with a slight wound, soon had delirium without fever, like an old toper; the fancies referred to the same subjects as in delirium potatorum, passed off under quieting treatment and opium, without maniacal attacks; after four days the delirium ceased, and the patient remained reasonable. Lastly, I must mention those rare and interesting cases where,

after operations in otherwise healthy persons, psychical disturbances develop, cases which evade all attempts at explanation, and are only analogous to cases where, after acute diseases, such as pneumonia, acute rheumatism, or typhus, the development of true mania is observed. In the Berlin surgical clinic I saw two such cases, in both of which, after total rhinoplasty, there was melancholy with religious hallucinations. Both patients were Catholic: one, a young man, incessantly worried himself trying to understand the idea of the Trinity; the other patient, a young woman, sought by prayers and castigations to atone for giving way to her vanity so far as to have a new nose made to replace the one lost by lupus. In the young man there were frequent outbursts of rage; both patients perfectly recovered after a few weeks. I have heard that *Von Langenbeck*, in Berlin, had another such case after a plastic operation, and *Von Gräfe* and *Esmarch* have had them after operations on the eyes. But these cases are very rare.

## APPENDIX TO CHAPTER XIII.

### POISONED WOUNDS.

We have still to treat of some varieties of injuries, where at the time of the injury poison is inoculated, which sometimes induces severe local symptoms, sometimes dangerous general disease. It is well known that these poisons are peculiar to some animals, and in others they develop as a result of certain diseases, and are then transferred by the diseased animal to man.

The results from punctures of a large number of small insects are scarcely in proportion to the slight mechanical irritation caused by their stings; it may, it is true, depend partly on peculiar susceptibility of the skin, if persons have extensive temporary inflammations of the skin after bites by bugs, midges, or fleas, while others are not affected by them. A needle-puncture is a much greater injury than a flea-bite, but the latter is followed by itching and burning, and the formation of wheals on the skin, while the results of the former amount to nothing. Hence it is not improbable that in the case of the wound made by the insect some irritating substance enters the skin. As is known, the stings of bees and wasps excite even greater disturbances; occasionally there is an extensive, very painful inflammation of the skin, with great redness and swelling, which usually terminates in resolution, and does not prove dangerous, but may be very annoying. A large number of such stings at the same time is not altogether free from danger; such stings on the tongue, in the palate, or on the eyelids, may from their locality cause certain dangers by the swelling induced. But, as these inflammations subside in a relatively short time, a physician is rarely called; the popular treatment is by various cooling remedies to alleviate the pain, among which I shall merely mention the application of moist clay, raw mashed potato, cabbage-leaves, etc. In more severe inflammations, lotions of lead-water and other antiphlogistic remedies may be resorted to. Still more severe than the stings of bees and wasps are those from *tarantulæ* and *scorpions*, that are seen in southern countries. They are followed by more extensive inflammation of the skin, with severe burning pains, occasionally by formation of vesicles; there may also be fever, but there is usually no danger, unless it arise from the locality of the injury. The treatment should be that above given.

Fortunately, with us there are few varieties of *poisonous serpents*, and even they are not frequent. Among them are the *Vipera Berus* (cross adder), and *Vipera Redii*, with two hook-like, curved fangs, containing the excretory ducts of small glands, which, at the time of the bite, pour their poison into the wound. The bite of these serpents is not so dangerous as is supposed; according to statistics, about two die out of sixty persons bitten. The pain is very severe; there are great inflammation, tension and swelling of the skin, with high fever, great anxiety, depression, vomiting, and occasionally slight icterus. The best treatment is to suck out the wound at once, as the poison is not absorbed by the gastric or oral mucous membrane. The wound should be washed at once, and it is advised to ligate the injured limb above the wound to prevent the absorption of the poison; but this has usually taken place by the time the patient reaches the surgeon; it is a disputed point whether the application of cups, the cauterization, burning or excision of the wound, be now of any service, but I should think its cauterization advisable. The local cutaneous inflammation is treated with special attention to the intense pain; by applications of oil, protecting the skin from the air by various remedies, with which we become acquainted in the treatment of superficial burns. Internally we usually give an emetic, then antiseptic remedies. Of all snake-bites in southern countries, those of the rattlesnake are most dangerous; sometimes they prove fatal in a few hours; the local inflammation of the skin, which is very severe and extensive, not unfrequently ends in gangrene; those bitten die with high fever, delirium, and sopor. [Prof. *Halford*, of Australia, treats snake-bites by injecting diluted liquor ammoniæ into the veins. See London *Medical Times and Gazette*, 1869, page 123.]

*Cadaveric poison* is a very phlogogenous substance, which probably varies greatly in its chemical composition. Some of you may have already had some experience on this point, in the dissecting-room. This putrid poison develops in the corpses of men and animals; in handling these, some of the juice from the dead tissue enters small, insignificant, and scarcely noticeable injuries of the skin, very disagreeable symptoms may develop. The resulting conditions are various, sometimes very malignant. Cases occur which were formerly seen particularly often in England, where at first there is little pain in the wound, but there are great depression, headache, fever, and nausea; then come delirium and sopor, and in some cases death takes place in forty hours. It is asserted that these worst cases of septicæmia were most frequent, from autopsies made soon after death, on bodies still warm, and it was doubtful if in these cases the surgeon had not inoculated himself with morbid matter developed in the body while still living, for the state usually termed putrefaction could not have begun. As a contrast to this malignant acute form, we may regard those cases where the poison has a purely local action. In the course of twenty-four hours there are moderate pain and slight induration in the injured finger; then a dry scab forms on the wound; under it there is always some pus. The scab forms as often as it is removed, the part remains painful and hard; in the course of time the epidermis thickens over it, and it forms a painful, wart-like nodule, moist on the surface. One inclined to this purely local development is usually less disposed to general infection. Between these two forms stands a third, where an inflammation of the lymphatic vessels and axillary glands accompanies the local inflammation; under early treatment this may end in resolution, but it often leads to abscesses in the arm.

For the first treatment of the part poisoned by cadaveric matter, I advise you to let cold water run on the wound for a long time, and not to check the bleeding, if there be any. In many cases the injurious matter will be at once washed out, and there will be no further infection. Should the parts around the wound redden, you may cauterize with nitrate of silver or fuming nitric acid; this is very painful, but it acts well; not unfrequently pus forms again under the resulting slough; in this case you remove the slough, and cauterize again, and repeat this till no pus forms under the slough.

Cauterization immediately after contact with the poison, from considerable experience on myself and on my students in the course on operations, I consider unadvisable. Small, lacerated wounds that do not bleed, and excoriations, are always more dangerous for infection than deeper incised wounds; the anatomical reason for this is that the lymphatic net-work lies chiefly in the most superficial layer of the cutis. Moreover, the susceptibility to the poison varies with the individual; repeated infections appear rather to increase than diminish the predisposition. Should lymphangitis begin, the arm should first of all be placed on a splint to keep it quiet, and then the treatment previously recommended for lymphangitis instituted. You may consider the course in the appearance of the above morbid symptoms to be as follows: A small quantity of liquid from the cadaver (or even of putrid pus from a living patient) is introduced into the wound; the lymphatic capillaries that have been opened take up this putrid matter and pass it into the trunks of the lymphatic vessels; coagulation may quickly take place here, and then the putrid matter acts as a specific irritant only on a small part; in other cases it acts on the lymph as a ferment, and the lymph coagulates in the next lymphatic glands, or else the swelling of the gland compresses the intra-glandular lymphatic vessels and so obstructs the passage through the gland; in this case also the disease remains local, although extending some distance, and not unfrequently leading to suppuration with fever (as in other non-specific inflammations). Lastly, the rarest cases: the fermented lymph, which even yet acts as a ferment, passes into the blood, and there excites chemical changes. Then we have a *septicæmia*, from cadaveric poison. From the cases that end in recovery we see that the injurious substances developed by the process may be again eliminated from the body by the secretions and excretions, but we do not know in what particular way this is done. In some cases some putrid substance is encapsulated in a lymphatic gland or other inflamed part, and may there lie harmless and after a time be gradually eliminated; but on active movement the poison may be again driven into the lymphatic vessels by the increased pressure of the blood, and there induce new, acute, local, and general infection. If indurated lymphatic glands remain after infection with cadaveric poison, daily warm baths are the best means for promoting the excretion of the poison.

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We have still to treat of some poisons which in certain diseases develop in animals, and may thence be transferred to man. Under this head come *glanders*, *carbuncle*, and *hydrophobia*.

Glanders (maliasmus, morve) is a disease which develops primarily in horses and asses. It is an inflammation of the nasal mucous membrane, in which this membrane becomes very thick, and secretes a thick, tough pus, and where, by the breaking down of caseous nodules, ulcers with a caseous base form; swellings of the lymphatic glands, occasionally tubercle-like nodules in the lungs, and acute marasmus, occur, and acute cases are usually fatal. The more chronic and milder form of glanders is called "farcy;" it is rarer, and gives a better prognosis. The glanders and farcy of animals are only conveyed to man by accidental inoculation. If some of the pus of a glandered horse enters a wound or excoriated spot on a man, or if very intense poisonous glander-pus fall on the uninjured skin at a point where the epidermis is thin, there may be very acute inflammation with general septicæmia, which in most cases proves fatal. The chronic form of glanders is rare in man; the symptoms are chiefly pustulous inflammations of the skin, and formation of abscesses at different points in the subcutaneous tissue; it is not so dangerous. In some cases of acute glander-poisoning there is lymphangitis and suppuration, limited to the injured extremity; in others a diffuse erysipelatous redness of the skin with great swelling develops quickly, while at the same time there is very intense fever. The local inflammation may go on to gangrene; there is delirium, and soon coma occurs; there may also be diarrhœa, purulent discharge from the nose, and pain in the muscles, with which symptoms the patient dies. The disease may run its course very rapidly; I remember, when a student in the Göttingen clinic, seeing a strong, robust man die of glanders in a few days; but patients with acute glanders may live from ten to fourteen days, and all the symptoms of pyæmia may develop in them, and numerous hæmorrhagic abscesses form in the muscles, which are so characteristic of glanders that they confirm the diagnosis. In rare cases acute, rapidly-fatal glanders may develop from the chronic; the reverse is also seen. Of course, persons that have much to do with horses are chiefly exposed to this disease, which never occurs primarily in man. Unfortunately, there is little hope from treatment in this disease; as in acute pyæmia, we treat the most prominent symptoms. Iodine, arsenic, and creosote, have been recommended as antidotes in glanders.

*Carbuncle* (anthrax, pustula maligna) is an infectious disease occurring primarily most often in cattle. It is called in German "Milzbrand" (gangrene of spleen), because in animals that have died of it the spleen is found greatly swollen, dark red, and gangrenous; in many cases also the intestinal mucous membrane is bloody-red and swollen; the loose subperitoneal cellular tissue, and occasionally the subcutaneous cellular tissue, of one of the limbs is often the seat of brawny infiltration; in the intestinal mucous membrane, and sometimes in the skin, carbunculous infiltrations may occur. As in all infectious diseases, the course varies in rapidity according to the amount and intensity of the poison absorbed and the resisting powers of the patient; it mav be *foudroyante* (apoplectiform), or may go on for several days. The herbivora are more readily infected than omnivora or carnivora. The contagion adheres to the products of the disease and the patient. Nothing certain is known about the origin; since it is more frequent in some regions than others, it has been thought that the soil and food had some effect. The intestinal secretions are mixed with the dung of the animals, and their poisonous effects have been proved. If such dung be spread over the land, and, either fresh or dried on hay, be eaten by other animals, they may be attacked by the disease.

Transfer of the affection to man is most often through the matter of the pustule; if this or the dried skin of the dead animal be brought in contact with the skin of man, even if it is uninjured, the poison may enter through a hair-follicle or sweat-gland; the result is a pustule, at first unnoticed, then itching and burning, in the centre of which a black blood-blister soon forms; high fever soon comes on. In bad cases the cutaneous inflammation early assumes the character of carbuncle, terminating quickly in gangrene, and if left to itself the disease is usually fatal.

Internally we give the ordinary antiseptics; the anthrax itself is to be energetically attacked by incision, excision, caustic potash, nitric acid, etc. If the patient comes under treatment early and there is no intense blood-poisoning, there is hope of cure; if the pustule is fully developed and septic symptoms have begun, death is certain. Recent observations show that infection in veterinary surgeons from post-mortem examinations of diseased animals does not have such a dangerous course, but often gives rise to a phlegmon of medium intensity, which may pass off in a few days with scaling off of the skin. Quite lately *Leube* and *W. Müller* have described cases where severe intestinal inflammation ending fatally followed the use of flesh from animals that had died of carbuncle. According to *Bollinger*, the milk of cows with this disease proves infectious to man. It is still a disputed question whether malignant pustule may also develop primarily in man; whether the malignant carbuncle described in Lecture XXI. always comes from infection, or may come spontaneously from the same causes as in animals. Eminent surgeons and veterinarians have investigated this subject; inoculations of secretion from malignant carbuncles of man on animals have proved very uncertain; observations have been contradictory; in short, the relation of these different forms of carbuncle and pustules to each other, in regard to their etiology, has not yet been cleared up.

Of late the view that the septic poison of carbuncle is associated with certain small organisms is gaining ground. *Davaine* especially holds that the bacteria (first described by *Pollender* in 1855) quite constantly found in the blood of living animals with carbuncle, or of those that have died of this disease, are the cause of the affection.

But we may doubt if it cannot exist without bacteria, as it is asserted that with blood from carbunculous animals, which contains no bacteria, other animals may be infected. In *Leube's* cases, already mentioned, countless cocci and bacteria were found in the intestinal mucous membrane (mykosis intestinalis, *Buhl*). Many assert that the bacteria found in carbuncle differ from those resulting from decomposition. *Bollinger* asserts that small cocci (bacteria germs) exist in the blood of every animal affected with carbuncle, but that from their small size they often escape observation; he considers their vegetation as the essential cause of the disease, which is, however, favored by the species of the animal, its nourishment, and the character of the soil and stabling. My own observations have shown me that the bacteria of carbuncle, like those in the blood and pericardium of decomposing bodies, belong to the meso- and megalo-bacteria; and also that cocci and even permanent germs (Dauersporen) often form in them. On inoculating blood containing bacteria in the cornea of rabbits, *Frisch* saw stellate figures form, evidently composed of bacteria, which developed enormously and led to suppuration of the eyeball, but never to general infection or the death of the animal. The inoculations of blood from horses and cattle which had shortly before died of carbuncle on rabbits, Guinea-pigs, sheep, and dogs, in the cases I witnessed, proved more certain in proportion to the certainty of the presence of bacteria; and uncertain results occurred, as they did in the cases of other observers.

We must also mention the mouth and hoof disease of cattle, as recent observations have proved its transfer to man. In cattle the disease consists in the formation of vesicles and pustules on the mucous membrane of the mouth, at the roots of the hoof, and on the udders of cows; these heal spontaneously in from five to fourteen days; and although the animals often emaciate greatly, only the young ones ever die. The disease seems to spread epidemically through the secretion from the pustules, the milk, and perhaps also through an evanescent contagion. The transfer of the affection to man results from contact of abrasions of the skin with the matter from the pustules, or from free use of *uncooked* milk of the diseased animals. If the latter has been the mode of origin, vesicles and pustules form in the mouth and on the hands and feet, as in the cattle. Catarrh of the throat and stomach may be added. The treatment consists in frequently rinsing the mouth, painting the vesicles with solution of borax (five parts to thirty of honey), and touching the pustules on hands and feet with nitrate of silver. Cooking destroys the infecting matter in the milk. It is not improbable that some aphthous diseases of small children arise from infection by milk thus diseased. In man the disease runs its course without much danger, as it does in cattle; only very young, feeble children could be endangered by it.

[According to *Letheby* ("Lectures on Food"), "Dr. Livingstone tells us that when the flesh of animals affected with pleuro-pneumonia is eaten in South Africa by either natives or Europeans, it invariably produces malignant carbuncle. He says, indeed, that the effects of this poison were often experienced by the missionaries who had eaten the meat, even when the presence of the disease was scarcely perceptible. . . . The virus, he says, is neither destroyed by boiling nor by roasting, and of this fact he had innumerable instances. Now it is a remarkable circumstance that ever since the importation of this disease (pleuro-pneumonia) into England from Holland in 1842, the annual number of deaths from carbuncle, phlegmon, and boils has been gradually increasing."]

*Canine madness* (hydrophobia, lyssa), which is transferred from animals to men, is better known and more frequent than either of the above diseases. From unknown reasons, the disease appears to develop primarily only in dogs; but from the bite of this animal, and the entrance of its saliva into the wound, it may be transferred to any animal, and apparently the poison does not decrease by inoculation, but is always propagated with equal power. For instance, a mad dog bites a cat; the disease develops in the latter, and she bites a man; an animal being inoculated with the saliva or blood of the man will have the disease.

The symptoms in the dog are described by the veterinarians as follows: We distinguish a raving and a quiet madness; previous to both of them the dog is downcast and eats little. After this state has lasted about a week the raving madness begins; the dog runs about in an objectless, unsteady way, apparently urged by some inward anxiety; if irritated, he bites at any thing coming in his way; the mouth is dry; he tries to drink, but soon runs from the water without taking it; he emaciates, he totters, then his hind-legs become paralyzed, his barking changes to a kind of howl, twitchings come on, and in three or four days are followed by death. In the still madness, paralysis of the muscles of the lower jaw occurs early, rendering biting and eating impossible. The other symptoms are the same as just described. Some do not consider these two forms of the disease as distinct, but as different stages, only lasting a longer or shorter time. On autopsy of animals dying from this disease, we usually find the gastric and intestinal mucous membrane much reddened; this is probably merely due to the various foreign bodies that the dog has swallowed. Beyond this, we find nothing abnormal, especially in the brain and spinal medulla; but we must add that hitherto no microscopical examinations of these parts have been made, while it is very probable that, in cases where paralysis very evidently occurs, there is degeneration of the spinal medulla, although otherwise the predominant character of the disease is humoral.

As regards the transfer of hydrophobic poison to man, it is a relief to know that all those bitten do not become sick, but that only about one out of twenty cases bitten is attacked. Usually the bite heals readily; more rarely it suppurates a long time, which is to be regarded as very favorable; the local reaction is never of such a nature as to threaten danger, and in this respect the hydrophobic poison differs essentially from the animal poisons heretofore mentioned; it is not a phlogogenous poison. The outbreak of the disease rarely occurs in less than six weeks after the bite, frequently even later; a case has recently been observed where the disease first appeared after six months. Older writers give a still longer period of incubation; there is a popular belief that the figure 9 plays an important *rôle;* it is said that the disease appears the 9th day, the 9th week, or the 9th month after the bite, and that before the end of the 9th year there is no security that the disease will not appear. This is certainly a fable, which is readily explained by the fact that the long duration of the incubation is very strange, and has given rise to the various stories. Where the poison remains hidden during this long time, whether in the cicatrix, in the next lymphatic glands, or in the blood, is entirely unknown. In a few cases only it has been observed that, shortly before the outbreak of the disease, the patient had noticed a slight redness of the cicatrix; then the first symptoms were great irritability, excitement, and restlessness, and in rare cases, even in this stage, there were spasms on attempting to swallow. The irritability constantly increases; the light, every noise or draught, pains these unfortunate patients, and may excite general spasms and the pains on swallowing. Now, very gradually, the fear of water appears; the patients suffer from unspeakable thirst, and as soon as they see any liquid they are attacked by horrible anxiety and spasms; occasionally, attacks of deep spasmodic inspiration follow; the patient cannot sleep, and is in constant dread of the least sound, as any thing excites the convulsions, which finally affect the whole body, and then lead to actual madness, with the appearance of most fearful anxiety. But, on the whole, the patients may be readily calmed by quiet and by speaking to them, and become either perfectly resigned or melancholy. Occasionally they warn those about them not to come too near or they may bite them, but they are not at all malignant, as they were formerly described. Great salivation and foaming from the mouth do not begin till toward the end; in some cases death is preceded by the severest tetanic spasms; others die after the convulsions and the fear of water have completely ceased, and when the patient and surgeon have been led into vain hopes. Unfortunately, pathological anatomy gives us no explanation of this wonderful and fearful disease. There can be no doubt that the spinal medulla is affected, but it has not yet been determined whether the nerve-substance itself is diseased.

As regards the prognosis, in those patients where the disease has broken out, there is no hope. It may be considered proper, in all cases, to cauterize or burn out the bites of mad animals, and to keep them suppurating a long time; at least this is the only rational treatment. It cannot be certainly decided from past observations whether excision of such a cicatrix can be useful after the disease has already broken out; it would at all events be a rational treatment. In the developed disease, almost all the powerful remedies in the materia medica and in surgery have been tried; all the narcotics have been used in large and small doses; opium and belladonna especially, used in almost poisonous doses, and the artificial benumbing of the patient, have at least alleviated their sufferings, if they have done no other good. The limb containing the cicatrix has been amputated in vain. In one patient, *Dieffenbach* tried transfusion in vain. Where there is dread of water, some fluid may be introduced through a tube; the patients are most comfortable when at absolute rest in a half-darkened room; in combating the convulsions, chloroform narcosis has repeatedly proved most serviceable, and patients who have once become acquainted with this remedy beg for it again. But this comprises the little that we can do for these unfortunates.

[Lately *Weller* claims that an anatomical characteristic of *Lyssa* in dogs, as in men, is a marked hyperæmia in the medulla oblongata and upper part of the cervical medulla with marked fulness of the perivascular spaces of lymphoid cells; this process differs from acute myelitis by not going on to softening.

The only treatment that offers any hope in the developed disease is by subcutaneous injections of *curare*. *Curare* paralyzes voluntary muscles, and, when given subcutaneously, repeated till relaxation results, may cure some cases; but its efficacy depends much on the special sample used. Of late, some cases have been arrested by stretching the chief nerve-trunk or some of its branches, in the limb primarily affected. We do not know what changes may be induced in the nerve, nor is it always successful, even when we know the point of origin of the tetanus. The fact of nerve-stretching having proved useful in some cases would tend to show that tetanus is due to some affection of the peripheral nerves. The number of deaths yearly from hydrophobia is, in Prussia, 71 ; Austria, 58 ; France, 24 ; Bavaria, 17 ; while in Constantinople, where there are so many dogs, it is said the disease does not occur.

Sometimes a single sting from an insect will cause severe symptoms of general poisoning, without our being able to suggest any other cause for such unusual reaction than individual predisposition, or, possibly, a direct entrance of the poison into a large lymphatic vessel. The patient quickly becomes comatose, the skin cool and covered with clammy sweat, the face cyanotic, breathing superficial and slow, the pulse rapid and scarcely perceptible. These symptoms usually subside in a few hours, along with the local symptoms ; but for some days the patients continue to feel heavy.

In Southern Germany, bee-tenders treat stings with scorpion-oil (olive-oil containing scorpions) ; sensible and trustworthy observers say this oil is a very decided antidote ; swelling and pain subside soon after its application. If this oil is not to be had, we may apply ammonia over the injured part, but this is only useful immediately after the sting.

All observations made with snake-poison show that its quality is always the same. The bites of poisonous serpents always cause the same series of symptoms, although the intensity of the action may vary with the variety of the serpent, and even with the same animal, according to the season of the year, and to the length of time that has elapsed since it last bit anything. If the bites follow each other quickly, the secretion from the poison-glands is exhausted, so that the later bites are less active than the first.

Even when dried the poison retains its activity, nor is it destroyed by alcohol ; for persons have been poisoned, even fatally, by injuring themselves with the fangs of serpents preserved in alcohol.

In the English provinces of the East Indies, as many as twenty thousand persons are said to die annually from snake-bites.

Poison in the secretion of certain inflamed parts, as puerperal endometritis, septic peritonitis, etc., may be very virulent. Infection from this source depends on the facility with which it passes through the skin ; small tears which do not bleed, and excoriations, are more susceptible to infection than deep incised wounds ; this is because in the latter case the flowing blood washes the putrid poison from the wound.

Dangerous infections with cadaveric poisons usually follow injuries so slight that they cannot afterward be found, and nothing is noticed till, a few hours after the infection, at the point of injury, there may be discoloration and swelling. In such cases there is only one remedy for combating the severe general symptoms, as for opposing the primary gangrene induced by contact with the cadaveric poison: this is a long, deep incision, dividing the discolored skin throughout its length, and so that its edges may gape; a cross-cut may also be required. Bleeding should be encouraged, and the wound may be sucked. Local cauterizations are not indicated—they cannot affect the virus already diffused in the tissue; but you may pack the wound with charpie soaked in *Burow's* fluid and envelop the limb in compresses wet in hot water, changing this every three hours. At the same time the limb should be kept immovable and elevated, and quiet secured by morphine.

Carbuncle-poison has been very carefully studied, and it is found to accompany a long, rod-like fungus, the bacillus anthracis; this is found in the blood and tissues of the carbunculous animal, especially in the vessels of the intestinal follicles and kidneys; it may be bred in suitable fluid, and this being introduced into the blood of healthy animals will cause carbuncle. Its resisting power is great; when dried the contagion probably lasts indefinitely; heating to 220° kills it, but it resists the cold induced by liquefying carbonic acid. The organized contagion clings to the products of the disease, and everything that has come in contact with the diseased animal may carry the germs. According to *Pasteur*, infection probably never occurs through the uninjured skin or the lungs, but chiefly through injuries of the mucous membrane of the digestive tract. In the parts of France where carbuncle is epidemic, the contagion from dying and slaughtered animals is passed to the vegetation and soil before the cadaver putrefies; putrefaction destroys the bacilli. Animals become infected from their food containing bacilli coming in contact with superficial injuries of the mucous membrane of the mouth and pharynx, which are very common, and may be demonstrated. The bacilli also occur in the soil; worms carry them about and bring them to the surface.

Inoculation of animals gives them a certain immunity to splenic fever. *Collin* has shown that the disease remains local till the poison enters the lymphatic glands, then it becomes general; *Toussaint* found that this generalization did not occur if inflammation was set up in the lymphatic glands by defibrinated blood of carbunculous animals from which the bacilli had been removed. If animals thus treated be inoculated with fresh poison, it will be re-

›y the glands, and the animal remains healthy. If pregnant e inoculated in the last months of pregnancy, their lambs ure against carbuncle. It seems as if all the physiological thological products might contain the poison ; even the milk ctious (*Bollinger*). All animals are not equally predisposed ; have entire immunity, in others the disease remains local. food, state of the soil, stabling, etc., have much to do with the emic occurrence of the disease. According to *Davaine*, the de-pment of carbuncle bacteria is most certainly arrested by tinc-e of iodine.

Many different plans of treatment have been tried ; besides in-rnal remedies, such as quinine, carbolic acid, iodine, etc., attempts ave always been made to destroy the poison that has entered the ody. Formerly, the infected point was removed by a crucial in-ision if possible, or several deep cuts were made, and the actual cautery, caustic potash, fuming nitric acid, chloride of zinc, etc., applied. The burning induced energetic reaction in the tissues, thus limiting the gangrene. Then subcutaneous injections of carbolic acid were tried, but they did not prove so successful as was hoped. Now many surgeons (*Roser* and others) recommend expectant treatment, moist warmth, lead-water, chloride of lime, etc., in cases where the extent of the local trouble indicates that neither excision nor cauterization would get rid of all the infiltrated tissue. Experience has shown that even extensive carbuncle with great loss of substance may heal spontaneously ; hence, it is well at the onset of the disease to incise and cauterize the diseased part, but, when the local redness and œdema are extensive, avoid excision and burning, and try expectant treatment and carbolic injections. The results of local treatment are best when the virus has not entered the lymphatic glands ; when the disease has become general, treatment is of little use, and the result depends on the resisting power of the patient. *Leube* and *W. Müller* have quite recently reported cases of fatal inflammation of the intestines after eating flesh of animals dying of carbuncle. In these cases the intestinal mucous membrane, especially the capillaries of the intestinal follicles, were found full of bacilli and cocci (mycosis intestinalis, *Buhl*). But the Gypsies in Hungary eat such flesh without fear or injury, while those who had butchered the animals had malignant pustules. Possibly the flesh was not eaten till it began to decompose, and the bacilli were killed by this act.

The attention of physicians and sanitarians has lately been directed to a malady called *rag-disease* occurring among operatives in paper-factories, particularly in those employed in sorting and picking, and thus exposed to dust from the rags. It seems proved that this disease is caused by carbuncle contagium, the rags having been exposed to this poison in the blood or secretions of the animal; and when dried and disturbed the dust floats off to poison the workmen by getting into any sores or entering their lungs.

It is not yet certain whether splenic fever in man is always due to infection, or may arise spontaneously under certain conditions.]

The three diseases last mentioned enter so much into the domain of veterinary surgery, sanitary regulations, and internal medicine, that I could here give you only a slight sketch of them. You will find more accurate information on the subject in *Virchow's* special pathology, Bd. II., Section Zoonosen, where the special literature is also given. In the surgery published by *V. Pitha* and myself you will also find (vol. i., part ii.) an exhaustive section on the Zoonoses.

# CHAPTER XIV.

## *CHRONIC INFLAMMATION, ESPECIALLY OF THE SOFT PARTS.*

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### LECTURE XXVIII.

**Anatomy: 1. Thickening, Hypertrophy; 2. Hypersecretion; 3. Suppuration, Cold Abscesses, Congestive Abscesses, Fistulæ, Ulceration.—Results of Chronic Inflammation.—General Symptomatology.—Course.**

GENTLEMEN: Having thus far attended almost exclusively to acute affections, we now come to the chronic, and first of all to chronic inflammation.

In chronic inflammation also, as in acute, there are chemical and morphological changes and nutritive disturbances of tissue; they are followed by softening and solution, or molecular disintegration, or extensive slowly-developing necrosis of tissue. To these processes are added dilatation of the vessels, exudation, and formation of new tissue. This combination may vary; chronic inflammation leads to very complicated appearances, according as one or other stage of the process remains more or less permanent, and according as there is disintegration, softening, or hardening of the tissue implicated, and as to the varied fate of the inflammatory neoplasia. Etiologically, the conditions in chronic inflammation are much more complicated; for there it is not merely a question about an irritation only once, as an injury or a burn, and their sequences, but we have, 1, to explain the cause of the inflammation; and, 2, why it assumes a chronic character. I shall first explain to you what anatomical changes take place in the tissues during chronic inflammation, in doing which, just as we did in acute inflammation, we shall here take the connective-tissue as the ordinary seat of the disease. Besides the distention and multiplication of the capillary vessels by formation of loops in acute inflammation, we found serous and plastic infiltration of the tissue to be the essential anatomical appearances. In chronic inflammation, distention of the capillary vessels, or fluxion, is a less prominent symptom, while the new formation of tissue and serous infiltration seem to play a more important *rôle*. The cell-infiltration of the tissue takes place in few cases, as it does in acute inflammation; but the individual cells often attain a rather more complete development. In this process of development the intercellular tissue changes; the connective-tissue filaments lose their tough filamentary consistency, the distensibility and elasticity of the subcutaneous tissue are impaired, and the consequence, as regards the coarser, palpable, and visible consequences, is that the tissue becomes more swollen and fatty, and less movable than normal. This is the first stage of every chronic inflammation. The course may vary as follows:

1. The tissue remains permanently in this state of serous, and, to some extent, plastic firm infiltration; skin and subcutaneous cellular tissue, articular capsule, tendons, ligaments, fasciæ—in short, all these connective-tissue constituents of the body which are in the above state—on section present a rather homogeneous, fatty appearance. In diseases of the joints and their vicinity we see this most frequently, and, as this swelling of the joint goes on without any reddening of the skin, it was formerly called *tumor albus*, a name which tells nothing of the nature of the process, but which, limited to certain forms of joint-disease, is practically serviceable. You may readily imagine that tissue which has been little altered may return from this stage of the disease to its normal state. The infiltrated serum is reabsorbed; the cells, which have newly entered the tissue or have newly formed there, partly become connective-tissue corpuscles, and are partly destroyed; the connective tissue itself returns to its former condition, and, if the state of affairs be not exactly as it was, it is nearly so; occasionally a state of cicatricial thickening remains; during the development of the chronic inflammation there may also have been small extravasations or escapes of red blood-cells through the walls of the vessels, from the increased pressure (according to *Cohnheim*); these change to a brownish-red pigment, which, when present in quantities, gives a yellowish or grayish color to the tissue that has been diseased. As a result of the continued excess of nutrient material, which sometimes flows to the diseased part in chronic inflammation, the tissue-elements may become larger and thicker; the whole tissue may increase; it passes into a state of *simple hypertrophy*. But sometimes the plastic (cellular) infiltration in chronic inflammation may attain a particularly high grade; from the infiltrated young cells new connective tissue forms in the old, so that the skin may be thickened to three or four times the normal extent; this deposit of new tissue of similar formation, in the old, is called *hyperplasia* by the pathological anatomists

When the thickening of the skin assumes a nodular form, it is usually termed *elephantiasis* in the most general sense of the term. Such hypertrophies and hyperplasias of the connective tissue, which may form in the course of a chronic inflammation, hardly ever recede entirely, but often remain in the same state, even when their causes have been removed.

2. If you imagine the chronic inflammation, so far as you at present know it, transferred to a mucous or serous membrane, you will acknowledge that the secretion cannot remain normal during the pathological changes which affect the tissue of these membranes. Usually it increases, there is *hypersecretion ;* chronic inflammation of a synovial or mucous membrane may evince itself chiefly by this hypersecretion.

Chronic catarrh of the mucous membranes may affect chiefly the epithelial or the connective-tissue layer or the glands of the membrane; in many cases all three suffer to an equal extent. The same is the case in the synovial membrane of the joints; some forms of chronic articular inflammation are chiefly noticeable from a very free secretion of a watery synovia; in others, there is more thickening of the synovial membrane, and but little increase of secretion.

3. Chronic inflammation may also be accompanied by suppuration, and its finer changes are just as in the acute disease, except that every thing is slower. For instance, suppose there is at some part of the body a collection of wandering cells with a formation of fluid intercellular substance; at the same time, of course, the tissue in which these cells are infiltrated dies, as always happens in circumscribed cell-proliferations. The tissue surrounding the spot first diseased is gradually infiltrated with cells; and it also goes on to form fluid cellular tissue with the character of pus; the infiltrated tissue is the more disposed to suppurate and break down when its vessels are little developed and do not supply sufficient qualitative and quantitative nutrient material to maintain the further development of the excessive cells. In abscess, a circumscribed cavity containing pus is thus formed, its walls are constantly being changed to pus, *suppurating.* All this takes place very gradually, and frequently the symptoms usually appearing in inflammation are wanting; often there is no pain, redness, or elevation of temperature, in the affected part, and usually there is no fever. Hence this variety of abscess, which comes on chronically, is called *cold abscess ;* for this chronic suppuration we use the term ulceration ("verschwärung"). We might also term the whole cavity containing pus a hollow ulcer ("hohlgeschwur"); but in common language this expression is applied chiefly to small cavities, while larger, slowly-forming ones are called cold abscesses. If you

examine the pus from such an abscess microscopically, you will find it rich in fine molecules, but rather poor in well-developed pus-cells. This is because the pus has long been enclosed in the body, and is changed by disintegration of the pus-cells to molecules, and by chemical decomposition; by the latter rich excretions of fat, especially of cholesterine crystals, are formed. The appearance of the pus to the naked eye is also changed by these metamorphoses, for it is usually thinner and clearer than in the acute disease, and has a disagreeable odor like fatty acids, and may contain fibrinous flocculi and shreds of necrosed tissue. Sometimes it is months or years before the suppuration of the walls of a cold abscess has gone so far as to cause perforation of the skin. In some cases it even happened that such an abscess has existed for years, that the ulceration of its walls finally stops, and the latter are transformed to a cicatricial capsule, and the pus is thus completely encapsulated. If we have opportunity to examine such an abscess, we find in it an emulsion-like fluid, occasionally containg crystalline fat, and sometimes without a trace of pus-cells, so that, from the appearances, we could hardly infer that the sac in question had been an abscess, if the whole previous course did not show it. Much more rarely, in the course of time, when the abscess has ceased to grow, there is reabsorption of the fluid, a cheesy pulp being left. If the abscess has perforated outwardly, the pus is evacuated, and, under otherwise favorable circumstances, there may be healing, as we shall soon describe. But, for this to occur, the ulceration on the inner wall of the abscess must cease, which generally only occurs when there is a sufficient development of vessels in the walls of the abscess; under their influence the inner surface of the abscess changes to a vigorous granulation-tissue, and then it condenses and atrophies to cicatricial tissue, and the opposite walls of the cavity unite, as in the healing of acute or hot abscesses; the pus escaping from the opened cavity grows less, and finally ceases altogether. Some time subsequently we may still feel the subcutaneous cicatrix of the abscess as a callous thickening; but, in the course of time, this also passes off, and the abscess-cicatrix again assumes the characteristics of ordinary connective tissue. I will now make you acquainted with a technical name used for those abscesses which do not originate at the points where first seen, but which have moved partly from sinking of the pus, partly from the ulceration having progressed chiefly in one direction. For instance, there may be suppuration along the anterior part of the spinal column, which, following the loose cellular connective tissue behind the peritonæum, and travelling along the sheath of the psoas muscle, finally appears as an abscess beneath Poupart's ligament. These and similar abscesses are called *conges* *tive abscesses.* The mode of healing above indicated does not take place with desirable rapidity, but, unfortunately, the general and local conditions are occasionally of such a nature that, after the evacuation of the pus, acute inflammation, with fever, attacks the abscess, and pyæmia or febrile marasmus comes on, or else, in spite of the evacuation of the pus, the chronic ulceration goes on slowly but steadily in the walls of the cavity. In such cases the openings of these large, often deeply-seated cavities continually pour out a thin, bad pus; the openings of such abscesses, whether of small or large diameters, are called *fistulæ.*

You may also imagine the above process of suppuration or ulceration as transferred to a surface or membrane; then we should have a flat or *open ulcer*, but, as this is an object of special and great practical importance, we must treat of it in an independent chapter.

4. Chronic inflammation may take another course very like suppuration, that is, caseous degeneration of the inflammatory neoplasia. Imagine, again, a great collection of young cells, and suppose, further, that in the centre this group undergoes molecular disintegration, and forms a cheesy pulp without separation of fluid intercellular substance. Plastic infiltration goes on slowly in the periphery of the caseous spot, by the collection of wandering cells, but the infiltrated tissue also passes into the caseous metamorphosis, and thus the central focus constantly increases. Here, also, as in suppuration, the failure of a vascularization keeping pace with the cell-formation is the local cause of the disintegration; here is a form of ulceration that may be termed "caseous ulceration" (a vascular, dry necrosis). When these yellow spots are found in the cadaver, it is often supposed that they correspond to a dried collection of pus, but this is not true, or, at least, very rarely so; most of these cheesy collections were from the first in miniature what they now are in gross, and were never fluid pus. It may very readily be proved experimentally that these caseous spots may proceed directly from the inflammatory new formation without suppuration. If, for instance, by introducing a foreign body (as a seton) into the subcutaneous tissue of a rabbit, you excite continued inflammation, in the course of a few days a yellow, cheesy mass forms around the foreign body; it is true this is the same for the rabbit as pus is for a man, but it was never fluid pus. There are also morbid processes in man in which, during chronic inflammation, this caseous transformation occurs instead of suppuration. In man, the further fate of these foci varies. If the process take place in a part not too far below the surface, it may, by advancing from within outward, cause perforation; the pulp is evacuated, and the cavity may gradually close as a cold abscess does. When this is the termination, it is usu-

lly accompanied by secondary softening of the mass, which is at first dry and cheesy, and this fluid pulp under the microscope is found to be composed almost entirely of molecular granules, some fat, shreds of tissue, and half-atrophied cells. The above process may be seen especially often in chronic inflammation of the lymphatic glands, but in them the spontaneous throwing off of the caseous deposit takes place very slowly, hence these fistulæ of lymphatic glands often remain stationary for months or years.

Another termination is for the caseous deposit to attain only a slight extent, then to atrophy entirely, and to take up such a quantity of lime-salts as to finally form a *chalky concrement*, which is concentrically enclosed by a cicatrix. But, as was stated, this only occurs in small caseous deposits.

5. There is still another form of chronic inflammation, which is accompanied by the deposit of a peculiar substance, the so-called lardaceous or amyloid, from the blood. But I shall not enter into this subject further, for this form of disease occurs chiefly in the internal organs, and hence has only an indirect interest for us.

First, as regards the results of chronic inflammation in a purely histological view, they vary. The cell-infiltration and the neoplastic process goes on chiefly in the connective tissue, and after its termination the final result is either a *restitutio ad integrum* or a cicatrix after the part has been destroyed by ulceration. When this process attacks muscles or nerves, the tissues suffer severely secondarily. The contractile substance in the muscle, as well as the axis-cylinder and medullary sheath of the nerve-filament, is not unfrequently destroyed by molecular disintegration or fatty degeneration, due to the disturbance of nutrition. Hence atrophy of the muscles and paralysis may result from chronic inflammation. How far the regenerative power of muscles and nerves goes under such circumstances is not decided. Molecular destruction and fatty degeneration may also occur without inflammation of the connective tissue enveloping the muscles and nerves. But I do not think we are justified in terming such a process of fatty disintegration of the protoplasm inflammation of the muscles and nerves, as has been done by *Virchow* in the muscles, at least, although it must be acknowledged that, in the great majority of cases, the appearance of fat-granules in the protoplasm may be regarded as the first expression of pathological (but not always retrogressive) processes in the body of the cell (*Stricker*). The fatty disintegration of tissue may be the result of inflammation, or may even accompany t; but to seek in it the nature of the inflammation, and to regard the atter as a disturber of nutrition to so wide an extent, does not seem o render it more comprehensible or of practical benefit. We regard every inflammation as accompanied by infiltration of the tissue with cells.

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After these general anatomical considerations, let us briefly run through the *symptoms of chronic inflammation*. They are the same as in acute inflammation, only they often come in a different order and in other combinations, and are usually less intense.

*Swelling* of the diseased part is usually the first noticeable symptom; it depends partly on serous, partly on plastic infiltration. The parts feel doughy, and at first quite firm; if an abscess forms, as may happen in the course of weeks or months, fluctuation gradually becomes more evident. We shall only perceive *redness* of the inflamed parts, when they lie on the surface, for, as the vessels are occasionally but little distended, it is not very intense or extensive. We may readily detect chronic inflammation of the nasal mucous membrane, or of the conjunctiva, by the swelling, redness, and increased secretion. Chronically inflamed skin gradually assumes a bluish or brownish-red color. But, if the inflamed parts lie deep, the skin is not discolored, and only becomes red when the deep chronic inflammation finally implicates the skin, as in the perforation of cold abscesses. *Pain* is one of the symptoms of chronic inflammation that varies most; in some very tedious cases it is entirely absent, but in other cases may be very severe, having a tearing, boring character, sometimes appearing spontaneously, at others only on pressure, or on merely touching the parts. The *functional disturbance* depends essentially on the pain and on the anatomical changes in the parts. *Heat*, the temperature appearing elevated when the hand is laid on the part, is not usually marked, or is very slight.

*Fever* is a symptom not necessarily pertaining to chronic inflammation; it usually appears only when the inflammation assumes an acute character, as not unfrequently occurs during its course, especially when the body has been much debilitated by long-continued suppuration. Then we have the so-called *hectic fever*, a febris continua, or simply remittent, with great differences in the morning and evening temperature of the body, a fever with steep curves. According to my idea, this hectic or consumptive fever results from continued absorption of the products of inflammation, especially of disintegration; hence it is most frequent and most intense from rapid breaking down of the inner walls of large abscesses, and in rapid progressive ulceration. This fever often runs its course with rapid emaciation, night-sweats, and diarrhœa. Few patients stand such chronic suppurative fever long; though I observed a boy fourteen years old, with a fistula remaining after resection of the head of the femur and general lardaceous disease, a whole year, during which he had a continued febris remittens; he finally died from general dropsy.

The *course* of chronic inflammation may be classed under two general heads. In the first case, even the commencement of the disease is indistinct, and can scarcely be stated with any certainty by the patient. Sometimes it is a swelling, a moderate pain, or a slight disturbance of function that has called attention to a morbid state. Cases which have begun so insidiously usually maintain this character in their further course. In other cases, the chronic inflammation is a remnant of an acute process; the chronic course is interrupted from time to time by acute attacks, with fever. We can say least that is definite about the *duration* of chronic inflammation in general, as this above all things depends on the exciting causes, to which we shall soon come. I only entreat you to bear in mind that chronic inflammation, like the acute, has a tendency to terminate, to have a typical end, for the new formation never goes beyond the development of certain characteristic metamorphoses of tissue, which lead to development of connective tissue, or of a cicatrix in some way, unless the diseased tissue is destroyed by disintegration. Why it is important to remember this will be clearer to you when we treat of the limitation of other new formations, such as actual tumors. Of course the new formation attains no typical end when its causes cannot be removed, or do not spontaneously disappear, and when organs are destroyed that are necessary to life, or when the strength is exhausted by suppuration.

## LECTURE XXIX.

General Etiology of Chronic Inflammation.—External Continued Irritation.—Causes in the Body.—Empirical Idea of Diathesis and Dyscrasia.—General Symptomatology and Treatment of Morbid Diatheses and Dyscrasiæ. 1. The Lymphatic Diathesis (Scrofula); 2. Tuberculous Dyscrasia (Tuberculosis); 3. The Arthritic Diathesis; 4. The Scorbutic Dyscrasia; 5. Syphilitic Dyscrasia.

To-day we come to one of the most important parts, not only of this section, but of all medicine, that is, to the *causes of chronic inflammation*. We saw how acute inflammation resulted from an irritant acting once, and varied according to the anatomical condition of the irritated part, and the nature and extent of the irritation, but that it ran a relatively short and typical course. Now we have to deal with inflammations that last several months or years; here there must be a continued cause, a long-acting irritation, or some abnormal reaction to simple irritation. These continued irritations may be of a purely local character; let us consider them for a moment. When small animals, like the itch-insect, take up their abode in the skin, as they dig burrows like a badger's in the superficial layers of the cutis, lay eggs, and there lead their laborious life, they cause constant irritation of the skin; to this is added the scratching, and a chronic inflammation of the skin is thus caused and kept up. If spores of fungus locate in the epidermis, and there begin to grow and to multiply to millions of small vegetable organisms, the skin will be placed in a state of continued irritation by these little foreigners; and various chronic cutaneous eruptions will result, such as favus, herpes tonsurans, pityriasis versicolor, etc. If a pressure or friction act moderately but continuously on the skin, it also is a chronic irritation, which is particularly apt to induce thickening of the part of skin affected. The callous spots on the heel and many corns are the result of the continued friction and pressure induced by our modern foot-coverings. In the same way the workman who uses axe and hammer a great deal has callosities in the hand, the shoemaker has them on the outer side of the little finger and hand where he daily draws on the pack-thread, etc. [We see the same thing much more markedly on the side of the left thumb and forefinger in plasterers, from holding their plaster-board; and at the upper and posterior part of the front leg of some horses, from lying on their iron shoes.] Sometimes foreign bodies in the tissue keep up a continued chronic irritation in the surrounding parts. Continued or often-repeated chemical irritation of the tissue may also induce chronic inflammation; for instance, chronic gastric catarrh may be caused by the repeated use of schnaps or strong liquors. Continued stagnation of blood and lymph, as well as their coagulation in the vessels, first induces hyperplasia of the walls of the vessels, and of the parts immediately around them, distention and tortuosity of the vessels, and thickening of the tissue; the skin of the leg is particularly exposed to this disease when there is any continued opposition to the escape of venous blood from the extremity.

When we have to treat chronic inflammations that may be traced to such external continued irritations, of which many more illustrations might be given, the results will be favorable. We get rid of the animal or vegetable parasites, the foreign bodies, the continued pressure, chemical influences, etc., and the chronic inflammation will disappear spontaneously. So far we have supposed a local irritation acting continuously on healthy tissue; if you suppose a tolerably severe irritation acting once on a tissue already diseased, you cannot expect the conditions to prove as favorable as in a simple traumatic inflammation of healthy tissue; but it is probable that the results, even of the single irritation, will be different, possibly more continued, because the conditions in the tissue will not be so favorable for typical removal of the disturbance. Suppose a portion of skin already suffering from chronic inflammation to be superficially contused, this single irritation may induce chronic suppuration, or even progressive ulceration, which, under normal conditions, would quickly have gone on to new formation of epidermis and healing.

The cases where we find such purely local causes for the origin and continuance of chronic inflammation are comparatively rare. In the great majority of cases the cause is not so evident; the case must be watched and tried in various ways before we can obtain any clew to the etiology of most chronic inflammations and diseases. We have not here mentioned miasm and contagion from the domain of general etiology; and we may leave them out of the question, for there is nothing to show that chronic inflammation may arise from a single action of contagion or miasm. It is true there are chronic malarial diseases, such as intermittents, etc.; but there the cause of injury acts continuously, and not unfrequently the disease can only be cured by removing the patient from the miasmatic atmosphere; hence this case corresponds to a continued external irritation. The same is true of repeatedly catching cold, where the new attack affects the body already diseased, and thus induces chronicity of the process. But all this does not suffice for the etiology of chronic inflammations; we must also look for the causes in certain congenital or developed conditions of the whole body. Let us hear what experience teaches on this subject.

On careful observation we first notice that certain forms of chronic inflammation constantly recur in certain organs and certain parts of the body; that at the same time they show themselves chiefly at certain ages and in persons presenting some similarities in their external conditions. Thus we see children of the same class, who are peculiarly disposed to chronic swelling and suppuration of the lymphatic glands, joints, and bones, other persons who are chiefly affected by insidious inflammation of the lungs, others who are particularly liable to colds and have pains in the different muscles and joints. We also see that such persons, who are constantly being attacked in the same way, transfer their individual pathological peculiarities to their descendants; that those leaving such legacies have in their turn received them from their fathers or mothers. To obtain some clear idea of individual morbid predispositions in this chaos, persons predisposed to certain chronic diseases were divided into groups; thus, in a purely empirical manner, men were divided, according to morbid dispositions or diatheses, into lymphatic, scrofulous, tuberculous, rheumatic, etc.

terms which at first merely meant that the scrofulous, for instance, were especially predisposed to glandular diseases; the tuberculous to the development of ulcerating nodules, etc. Subsequently this grouping was carried further, and it was concluded that a certain morbid condition of the physiological processes of the entire body must lie at the root of such predispositions. A morbid material, or essence, a materia peccans, was supposed to exist in the body; the most natural bearer of this was the blood, for this passed through the entire body, and its condition certainly gave a measure for the more or less normal or pathological condition of the entire body. The word dyscrasia (a bad mixture) indicated such a pathological condition of the blood; hence a scrofulous, tuberculous, etc., dyscrasia were spoken of. It is, however, a strange idea to burden the blood alone with the pathological changes of the whole body, and assume, as it were, that infection of the whole body resulted from it. This could only be acknowledged in cases where an abnormal material was introduced into the blood from without, as we have seen to be the case in poisoned wounds. But this is not the case in the dyscrasiæ under consideration, or at least it is only partially so; but the morbid dispositions develop in the body itself from causes little known, if they be not handed down as an inheritance from the parents. The blood is no more absolutely stable than any other tissue of the body; it is constantly being renewed, partly used up and again renewed, etc.; we do not certainly know the source for the renewal of the blood-corpuscles; you know from physiology that the serum of the blood is constantly being regenerated from the lymph, and this again from the chyle-vessels of the intestines, and you also know that fluid constituents from the blood are excreted by kidneys, lungs, and skin. How little we know of these things, and how complicated even these little affairs are! I lead you to this consideration to add that normal blood can only form from a healthy body, and the reverse; hence that we cannot physiologically speak of a one-sided disease of the blood. But there would be no use waging war against and trying to root out the words dyscrasia and diathesis, now firmly embedded in medical language. It would do science no harm to use them forever with the above meaning; we must have a name for these things, for they are not myths, but are facts that have been observed for centuries, although their significance has varied greatly. We may go too far in classifying persons in this matter, if we ascribe to every one a pathological diathesis, or try to place every patient in one of the chief divisions. Although there might theoretically be a certain amount of correctness in supposing that in our present state of cultivation there was no such thing as an absolutely healthy man, still, it would be very senseless to try to maintain this in practice. And you must not suppose that it is always so easy to class every patient in certain groups, just as plants are analyzed and their systems determined, for all classes of men may breed with each other; moreover, some abnormally-formed individuals may become perfectly normal in the course of time, and the reverse; thus a number of middle forms naturally result, which defy any classification. There are now, as there have at all times been, physicians who are too skeptical about the existence of a general morbid disposition to certain forms of disease, and only acknowledge local and partly only accidental irritations as causes. Such a hyperskeptical current ran through modern medicine a short time since, and was perfectly justified, for the crasis doctrine had become so luxuriant, that there was scarcely a variety of inflammation, scarcely a disease, in fact, which was not based on some specific crasis. Whoever observes independently and carefully, and at the same time has the opportunity of seeing a variety of patients, will certainly arrive at the correct view in the course of time, and will neither throw himself too unreservedly into the arms of the crasis theory, nor set aside, as illusions and deceptions, the experiences of centuries. It is a question whether it be of any practical value to use such terms as scrofulous or syphilitic inflammation, if it would not be better to regard the chronic inflammatory processes without any regard to their origin. The future will decide this question; at present I deem it my duty as teacher to clear your views on these points as much as possible, and to place you in a position to be able to understand all your colleagues speaking on these subjects, no matter to what school they belong. But enough of this general explanation; let us draw a brief sketch of the different diatheses and dyscrasiæ:

1. *The lymphatic or scrofulous diathesis* (*scrofula*). This tendency to disease exists chiefly during childhood, though more advanced ages are not free from it. Persons with this diathesis, especially children, are greatly disposed to chronic inflammatory swellings of the lymphatic glands, even after inconsiderable irritations, to certain inflammations of the skin (eczema, impetigo), especially of the face and head, to catarrhal inflammations of the mucous membranes, especially of the conjunctiva, more rarely of the intestinal canal and respiratory organs, to chronic inflammations of the periosteum and of the synovial membranes of the joints. As regards the swelling of the lymphatic glands, especially of the submaxillary and occipital, it has been asserted that it is merely a result of irritation from dentition, or of the eczematous eruptions on the head, of the inflammations of the eye, ear, etc.; this is partly correct, but even taking this view, that all swellings of the lymphatic glands are secondary, even then for the

glands to swell after dentition, for instance, there must be an abnormal irritability of the lymphatic system such as does not exist in all children; moreover, such local irritations cannot always be found for the affections of the bronchial and mesenteric glands, which are almost as frequent. It is also a morbid state for the swellings of the lymphatic glands to last longer than the irritation, and even subsequently to increase without apparent cause. It may be acknowledged that some of the above affections—for instance, part of the scrofulous diseases of the joints—are caused by injuries, contusions, etc.; but the fact that they take a chronic and to some extent entirely peculiar, constant course, is due to abnormal condition of the tissue, which abnormal condition is so spread over the entire body that it cannot be regarded as a purely local, but must be considered a universal condition. Various attempts have been made to explain this local and general abnormity, especially to refer the "chronicity" to a continuance of the irritation, so as to escape the enigma of an organism reacting differently to one irritant from what it does to another. Hence it has been assumed that the matters formed by a chemical change in the tissues, from whatever cause, were not taken up by the lymph- and blood-vessels and removed from the diseased organ, but remained there and induced continued inflammatory irritation. I am far from denying that this takes place occasionally; but even if it were always true, the peculiarity just mentioned of this or that organ still remains abnormal in these persons. In short, we do not thus escape the fact that these persons differ from the majority either in certain tissues or *in toto*. Children fall times without number on knee, hip, or elbow, without any disease resulting, or else the effects pass off in a few days, even without treatment and when there has been considerable bruising, as shown by the extensive extravasation, swelling, and pain. But even after slight injuries some children have chronic inflammations of the joints; these are exceptions; there is, however, no objection to regarding them as a peculiar pathological race. Attempts have been made to diagnose the scrofulous diathesis from the general appearance and condition of the child. The following is the picture usually drawn of a scrofulous child: blond hair, blue eyes, very white skin with thick cellular membrane, thick lips, pot-belly, voracious appetite, and tendency to constipation (*torpid* scrofula). In practice you will meet some of the originals of this portrait, but you will see many other cases not at all like it, which nevertheless suffer from typical scrofula. I do not attach much importance to these external symptoms. In regard to the course and terminations of chronic inflammation in scrofulous children, we may make the following remarks: In a few cases the chronic inflammatory swelling sooner or later subsides entirely, and the parts become perfectly normal. The course with suppuration is the most frequent, and according to the special nature of the case this may be quite acute, as it is in inflammation of the submaxillary glands and in inflammations of the joints. Often the disease remains chronic for years; abscesses, fistulæ, ulcers, etc., form. Early suppuration occurs, especially in somewhat emaciated, debilitated, badly-nourished children, who are very liable to fever (*erethitic* scrofula), and its prognosis is very bad. The termination of the inflammation in caseous degeneration is not rare; it is particularly frequent in the lymphatic glands; of course it must have a very bad effect on the general nutrition, when the mesenteric glands are degenerated in this way, and the chyle-ducts thus mostly obstructed; incurable atrophy of the entire body may thus be induced. The lymphatic diathesis is in most cases congenital, and is transmitted from generation to generation; but it may also be developed by improper modes of life. Among the most injurious causes are given: chief or exclusive diet of potatoes, flour, or sour bread; unhealthy, damp dwellings; lack of cleanliness, fresh air, etc. It is indeed difficult to prove if all this be correct; at all events, if the above causes *always* induced scrofula, it would be much more frequent than it now is among the poor.

To state in a few words what is at present understood by a lymphatic constitution or scrofula, it may be considered—1, as a disposition to chronic inflammation of the skin, bones, and joints, in which the inflammation may lead to development of granulations, of pus, and to caseous degeneration; 2, as existing when swellings of the lymphatic glands, even when induced by temporary irritation, continue long in the same state, or even increase without new peripheral irritation.

We shall here pass at once to the *treatment* of scrofula in general. First of all, the diet should be regulated; good animal food, eggs, and milk, well-baked wheaten bread, occasional baths, residence in fresh, healthy air, a hardening mode of life, are the most important remedies, but from the circumstances they are often the most difficult to employ; in prescribing the diet, special attention must often be paid to the individual case, especially as to whether there is a tendency to lardaceous disease or atrophy, whether the digestive organs are normal, or were ruined in youth by improper diet. As the disease is very common among the poor (without the rich being free from it, however), these dietetic and hygienic rules are particularly difficult to follow. The number of internal anti-scrofulous remedies is very great; the object is not, as was formerly supposed, to introduce a specific remedy as an antidote to some unknown poison circulating in the blood, for the latter does not exist; but the treatment should be purely symptomatic, and usually general. From the above, you see that scrofula is not a materia peccans in the blood, but only a debility of the organization in some direction, a more or less intense predisposition to peculiar forms of disease. This is a decided difference from, and an advance beyond, the old view of the disease. From my explanation you may also understand those recent skeptics who think that all chronic inflammations in children are of similar origin, and that it is consequently unnecessary in each case of chronic inflammation of the lymphatic glands, or in articular inflammation, to add that it is scrofulous or depends on a lymphatic diathesis. Possibly these expressions may disappear in the course of time, as they will be rendered unnecessary by greater clearness of ideas, but it is not correct to say that all chronic inflammations in children have the same origin, for some of them may be due to hereditary or developed syphilis; and in adults there are many other constitutional predispositions besides those that have hitherto been termed scrofulous or tuberculous, and which consist in the predisposition to chronic inflammations ending in suppuration, caseous degeneration, and ulceration. It seems to me that there can be no doubt that these processes are, to a certain extent, opposed to other forms of chronic inflammation—for instance, to those depending on interstitial proliferation of connective tissue (cirrhosis of the liver, morbus Brightii, gray degeneration of the medulla spinalis, etc.).

Many things have been tried to improve the lymphatic diathesis. Formerly purgatives were occasionally given, and in England particularly small doses of mercury were administered; this is well suited to fat scrofulous children; burnt sponge, folia juglandis regiæ, herba jacea, acorn-coffee, and bitter medicines, were recommended, and are still used. At present, cod-liver oil is most used as an anti-scrofuletic, as it is not only considered to have a specific action against the scrofulous diathesis, but is very properly prized as exceedingly nutritious, and hence is especially used in emaciated scrofulous children; in fat children it might even prove injurious. Some of the preparations of iodine act very well in scrofula; but they should be employed carefully, and in fat rather than in atrophic children; iodide of iron is best in pale fat children, with fungous inflammations of the joints. The easily-digested preparations of iron are very valuable remedies in scrofula patients with anæmia. Salt-water baths also act beneficially; these may either be used at the springs, in Germany, for instance, at Kreuznach, Rheme, Wittekind, Coblenz, Tölz, Reichenhall; in Austria, at Hall, Ischl; in Switzerland, at Rheinfelden, Schweizerhall, Lavey, or Bex; or, they may be prepared at home by adding from, according to the size of the bath, one to three pounds of salt to a warm bath. For a large child, sea-baths may be recommended; for weakly children, warm baths with the addition of malt and aromatic herbs. In fat scrofulous children, *Niemeyer* recommends wrapping the whole body in wet sheets; I have seen good results from this in some cases. Some physicians also recommend sulphur-springs, especially the hot ones, in scrofulous diseases of the joints; so far, I have seen more harm than good from them. You see there is no lack of remedies; still we rarely succeed in improving the constitution by them, and cannot prevent relapses in all cases. Sometimes, too, the local process attains such a grade as to be of itself dangerous to life, and the local remedies must be mostly relied on. As before stated, the tendency to these diseases greatly decreases in the course of years; but many children die of the diseases of the bones and joints.

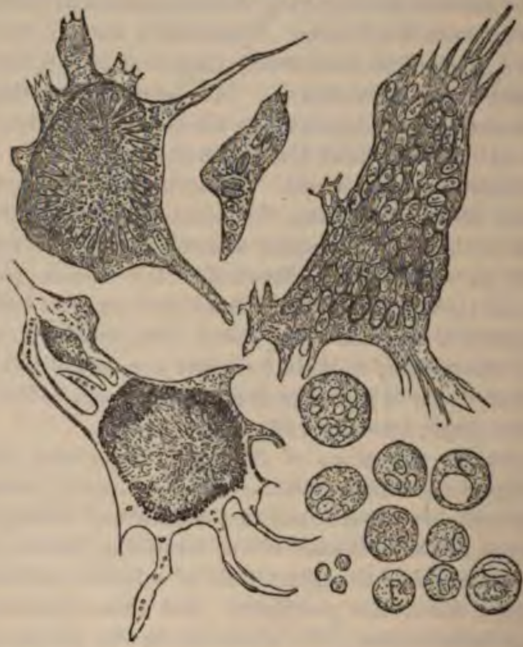
2. *The tuberculous dyscrasia. Tuberculosis.* The name of this disease comes from *tuberculum*, the nodule, because chronic inflammations due to this disease appear as small nodules, or tubercles, at first scarcely as large as a millet-seed, often microscopic. If you analyze one of these nodules with the microscope, you find it to consist of a number of medium-sized, round cells, which increase in the periphery of the nodule, while in its midst the short-lived cells have already broken down to a fine, molecular, dry pulp, which, when the nodule is very large, becomes yellow and caseous.

The recent investigations of *Schüppel*, *Langhans*, *Rindfleisch*, and others agree that large multinucleated masses of protoplasm, so-called giant cells, are often found in the centre of young tubercles; we shall speak of these further when describing the new formation of bone. The nuclei in the giant cells of tubercle are often exquisitely arranged about the periphery. But these giant cells do not always occur in tubercles. We often see in the peritoneum an indiscriminate grouping of large and small cells as a commencement of tubercle; and near these distinctly round or very irregular but sharply-bounded new formations there are more diffuse (tuberculous) infiltrations, which can scarcely be distinguished from ordinary inflammatory infiltration, except by the fact that the cells are nearly double the size of wandering cells which form the first cellular infiltration in acute inflammation.

A great peculiarity, especially noticed by *Rindfleisch*, is that tubercle often develops on and in the walls of small arteries and lymphatics, but very rarely in veins.

There are various views about the origin of the cells which form tubercles. If they are wandering cells, they must enlarge very rapidly soon after their escape from the capillaries and veins; on the whole, modern observers are little inclined to this view. *Rindfleisch*, *Kundrat*, and others hold that tubercle-cells develop mostly from proliferation of endothelium, especially that of the blood-vessels, lymphatics, and serous membranes. *Rindfleisch* thinks they may also develop from the muscle-cells of the arteries; *Ziegler* has proved that they may result from confluence of wandering cells.

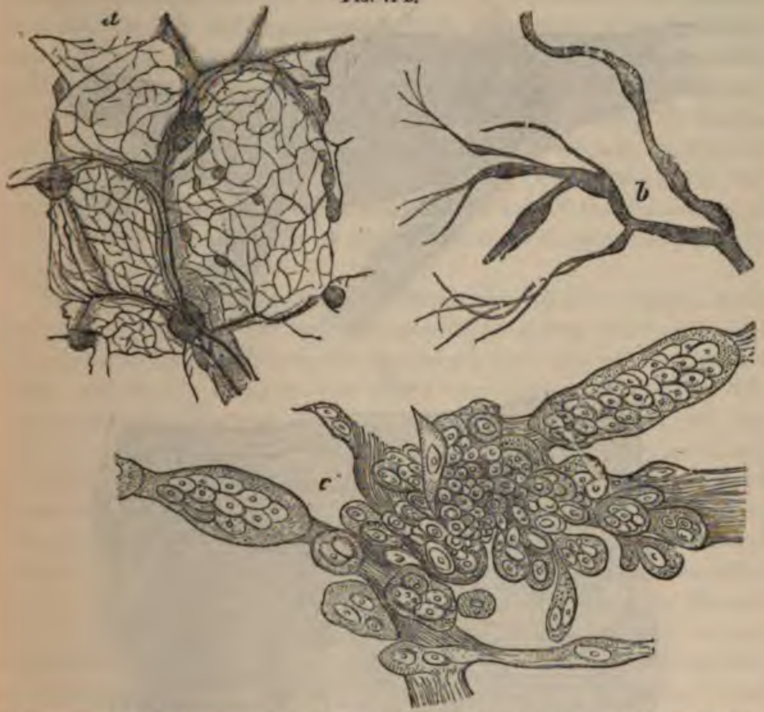
FIG. 71 A.



Giant cells from tubercle in various stages of development. After *Langhans*. Magnified about 400.

Regarding the subsequent fate of these small neoplasia, the most essential and peculiar thing about them is that vessels do not develop in them any more than in purely epithelial neoplasia, although their periphery is very vascular. Very rarely cases occur where the tubercles gradually become filament-nodules. While every other neoplasm is accompanied by growth of vessels, in tubercles this is wanting entirely, as has been lately shown again by *Rindfleisch*, *Heitzmann*, and others. The result of this is that the young neoplasia cannot live long; it dies in the centre, but the periphery survives. The dead centre occasionally breaks down into a fine, punctate, amorphous substance, which to the naked eye appears as a

FIG. 71 B.

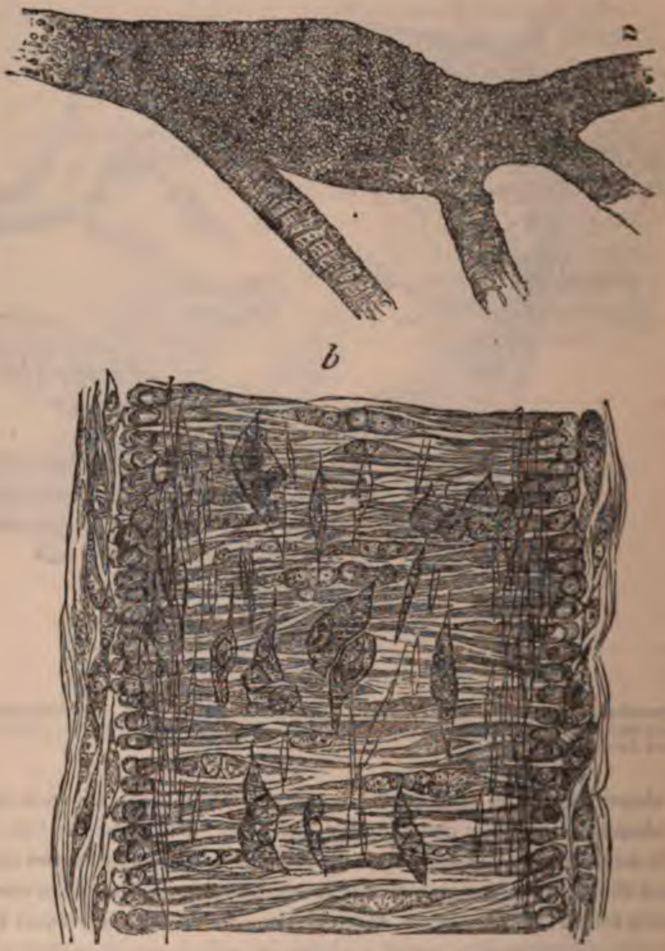


*a*, Minute tubercles in the peritoneum. *b*, Minute tubercles on a cerebral artery. *a* and *b* slightly magnified from preparations of *Rindfleisch*. c, Development of minute tubercles in the peritoneum. After *Kundrat*. Magnified 500.

dry, cheesy pulp ; in short, as a result of its lack of blood-vessels, the tubercle undergoes cheesy degeneration. Possibly the tubercle might enlarge *ad infinitum* by new cellular infiltration of the tissue around the primary focus, but this rarely happens. The large cheesy deposits found in the brain, testicle, etc., in most cases result from confluence of numerous small nodules, of which we often find numbers in the vicinity of large caseous nodules.

This brings us to the relation of the tissue to the tubercle scattered through it. I would here remark that the miliary nodules usually appear in large numbers in the organ or part affected. Just around the tubercle there is generally a subacute inflammation with free cell-infiltration and vascularization ; this may lead to suppurative softening of the tissues, chronic abscesses, and ulceration ; thus a cavity is formed which contains pus, softened shreds of tissue, and caseous tubercle. The inflamed parts around the tubercle may be drawn into the caseous degeneration, and a large cheesy deposit

Fig. 71 c.



a, Minute tubercle of a cerebral artery. Magnified 100. b, Commencement of the cellular growth one of the small cerebral arteries. Magnified about 1,000. (I do not think it can be proved wheth the multinucleated cells are wandering connective-tissue, endothelial, or muscle cells, or wheth they are due to the transformation of the intima to protoplasm.) Both drawings are preparations of *Rindfleisch*.

forms, which shall contain the primary tubercle; this may su quently soften by peripheral suppuration, or after encapsulation become calcareous. If tubercles form in mucous membranes, the larynx, intestine, ureters, bladder, or uterus, besides the culous infiltrations and ulcerations there is purulent catarr free detachment of epithelium, especially in the pulmonary (desquamative pneumonia, *Buhl*). In all of these cases the diseased part may be, but unfortunately rarely is, encapsulated by firm connective tissue, after undergoing metamorphosis; and after evacuation or calcification of the contents, the capsule may shrink to a firm cicatrix. But in serous membranes, and especially in the peritoneum, the inflammation caused by presence of tubercles leads at once to development of connective tissue, which not only encapsulates the nodules, but causes such an intimate adhesion of the intestines to each other and to the walls of the abdomen, that they can scarcely be separated on autopsy.

As regards the occurrence of tubercles in different organs, none are exempt, though some are more predisposed than others. Tubercles are most frequently found in the lungs, especially at their apices; there are usually many at one time; they unite, the walls of the bronchi are implicated in the process, they are destroyed, and the caseous, partially-softened contents of the tubercles are coughed up; sometimes blood-vessels are ruptured, giving rise to spitting of blood or pulmonary hæmorrhage. A space thus left by softened tubercle is called a *cavity*. It is not our object to enter more into detail; you will hereafter learn enough of this unhappy disease in the clinic. Next to the lungs, the most frequent location of the disease is in the laryngeal mucous membrane, then in the intestinal mucous membrane, even in the rectum, where the tuberculous ulcers and abscesses also acquire a surgical interest. Tubercles also occur in the bones, especially in the spongy ones, such as the calcaneus, bodies of the vertebræ, and upper epiphyses of the tibia. Although the lymphatic glands are often diseased in tuberculosis, miliary tubercle proper is hardly ever seen in them; still *Schüppel* found them there also.

The views as to the etiology of tuberculosis have changed wonderfully of late years. Formerly it was not doubted that it was partly an idiopathic disease, partly due to hereditary predisposition. Hence we spoke of a tuberculous as we did of a scrofulous diathesis, and the two were considered as related, although not identical. *Laennec* started the view that the small nodular neoplasiæ (gray miliary tubercles) were the primary development, and by confluence and growth led to the destruction of the affected tissues. The division of tubercles into miliary gray points and into cheesy nodules, the very peculiar acute miliary tuberculosis, the connection of tuberculosis with other and especially with chronic suppurative inflammations and those tending to caseous degeneration, were gradually developed and in many places remain obscure, although the idea of tubercle has been rendered more limited and precise by *Virchow*, so that at present every new formation that has undergone caseous degeneration is not considered as tubercle. It was reserved for *Buhl*, by careful experiments, to arrive at the idea that acute miliary tuberculosis was the proper type of tuberculous disease; he found it mostly combined with old caseous or purulent inflammatory foci; he made the bold assertion that it always resulted from absorption of substances from these foci. According to this, tuberculosis was an infectious disease, a sort of nodular exanthema on and in internal organs, caused by the absorption of an injurious substance, particularly from old caseous points of inflammation in the lymphatic glands, lungs, bone, etc., and some of these particles may have a specific infectious action, as emboli in the lymph- and blood-vessels. Investigations of late years have shown that many destructions—in the lungs, for instance—which previously had been considered due to miliary tuberculosis as a matter of course, are inspissated, caseous, and partly-softened spots, that must be regarded as the result of a simple chronic, ulcerative inflammation, as no miliary tubercles are found in them, but only large-celled infiltration. It seems, indeed, that even in pulmonary tuberculosis the formation of true tubercle is to be regarded as secondary and frequent, but by no means necessary. *Niemeyer* deserves great credit for his practical application of this view, according to which *a diathesis to chronic purulent inflammations of certain organs, but not the tuberculous infection, would be congenital.* This view is of late greatly supported by the fact that attempts to render animals, especially Guinea-pigs and rabbits, tuberculous, have succeeded. In these little animals irritation of very short duration excites inflammation with caseous purulent products, and from this focus results a tuberculous dyscrasia, which evinces itself in the production partly of miliary tubercles, especially on the serous membranes, partly of yellow nodules in the lung, liver, spleen, etc., and causes death. These very interesting experiments, which were begun by *Villemin*, and repeated by *Lebert* and *Wyss*, *Fox*, *Klebs*, *Cohnheim*, *Waldenburg*, *Menzel*, and others, with the same result, but with different interpretations, seem to me to prove, what I have always maintained, that tubercle is merely a peculiar form of inflammatory new formation; that is, that *Buhl's* view is correct. But it is important to remember that these inoculations only succeeded in animals having a tendency to cheesy degeneration, as rabbits, etc. *Rindfleisch* says these animals become tuberculous whenever they have a chronic inflammation. In dogs the inoculation does not succeed.

[The tendency for patients with points of cheesy degeneration to become tuberculous, explains the presence of young miliary tubercles in the lungs of patients with advanced phthisis; the latter occasioned the former. A long step in the etiology of phthisis was made when *Villemin*, from numerous experiments, declared the transmission of tuberculosis from one person to another; he said if tuberculous matter was introduced into the body of an animal, it acquired true tuberculosis. It made no difference if the introduction was by inoculation, injection, or feeding; it was only important that the tuberculous matter should be fresh and undecomposed.

It is only of late that the numerous apparent contradictions in the results of different experimenters have been explained. First it was shown that the introduction of indifferent bodies only induced tuberculosis when the animals were at the same time exposed to other opportunities for infection with tubercle. It was found that rabbits became tuberculous in the laboratories where these experiments were made, even when nothing was done to them; when not in the laboratory, tuberculization did not follow inoculation with indifferent bodies, and they could be inoculated with all kinds of caseous inflammatory products without showing signs of tuberculosis, but that certain pathological products invariably produced it. It makes no difference whether for this purpose we use a piece of tuberculous peritonæum, pia mater, or cheesy portion of lung, testicle, or lymphatic gland; always within a short time, according to *Cohnheim* and *Salomonsen*, in rabbits the twenty-first day, in Guinea-pigs a week earlier, there was an eruption of tubercles, when the inoculation was made from man to the animal or from one animal to another.

From these undoubted results of experiments tuberculosis must be regarded as a peculiarly infectious disease; all caseous products do not induce it, only those are active that had previously been regarded as products of tuberculous neoplasia, but are now considered the cause. Hence the views held originally by *Laennec* and *Rokitansky* as to the causal relation of acute miliary tuberculosis and tuberculous infiltration, again took position. Tuberculosis is excited by the transfer of tuberculous matter, and by nothing else.

Observation showed that not only specific tuberculous products, as sputa, infiltrated tissue, etc., proved active, but investigation of tuberculosis in cattle (so-called *Perlsucht*) showed that feeding on the milk of cows with this disease could transfer it to other animals; or that living in the same stall with them would communicate the disease. *Villemin* has found that dry, dusty tuberculous matter remained active, so it was natural to suppose that tuberculosis in man could also be induced by a material and probably dust-like contagion, and, from the great frequency of the primary disease in the lungs, that they were the most common way for the infection to enter the body; hence *Cohnheim* called it an inhalation-disease, although the virus may be taken in by the intestines and other ways.

Transfer of the disease from man to animals, dogs, rabbits, etc., by inhalation of finely-divided products, has been proved by numerous cases from acute miliary tubercle, tuberculous sputa, and the products of cheesy metamorphoses from the lungs and lymph-glands, and granulations from scrofulous inflammations of the bones and joints. So you see *Rokitansky* was correct when, in his fundamental work, he described yellow tubercles, cheesy metamorphosis, tuberculous infiltration, tuberculosis of the bones and synovial membranes, as results of tuberculosis, and treated scrofulous inflammation of the lymphatic glands as tuberculosis of these organs.

The infectious nature of tuberculosis once proved, the next question was as to the nature of tuberculous virus. *Hueter* and *Klebs* had already advanced the view that it was a fungus; of late, cocci have been found in the products, "monas tuberculosum" has been cultivated, and its inoculation is said to induce the same effects as those from tuberculous products.]

If from what has just been said we recognize to the full extent the immense progress recently made in the knowledge of tuberculosis, still we must not fail to see that it does not fully explain the interesting connection between some chronic surgical diseases and tuberculosis of internal organs, especially of the lungs. Although there are a good many cases where pulmonary tubercles follow chronic suppuration of bones or joints, and caseous degeneration of swollen lymphatic glands, just as often death of the patient results, after years of illness, from exhaustion, and on section we do not find a trace of tubercle. Under some circumstances, too, there is no absorption of the caseous masses, or else, if absorbed, they do not induce tubercle. This would go to prove that there must not only be a disposition of inflammatory foci to become caseous, but also a disposition to the dissemination of tubercles, and that these two dispositions are not necessarily combined as in the rabbit and Guinea-pig. The fact that around a small inoculation a cheesy focus forms, and from this disease is disseminated to the internal organs, is a peculiarity of these animals, as it is of some human beings. This peculiarity is called the tuberculous diathesis. Nor must I hide from you that some pathologists only acknowledge a frequent coincidence between chronic suppurating or caseous foci and tubercle, and refer both to a common unknown cause. But all this cannot prevent me from recognizing the exceeding value of the above-described recent observations, and regarding them as one of the greatest advances of modern pathology.

[Modern views of tuberculosis have affected the *treatment;* firstly, as to prophylaxis, knowing that it may not only be derived from men, but also from beasts. But the surgeon is especially interested in the extension from local foci: we do not know how long it may require for a general infection to result from a local affection; we cannot always decide whether an external tuberculous focus is the result of a primary local infection, or whether there has been tuberculosis in some other part of the body that has escaped our observation. *Cohnheim* says the bronchial and tracheal lymphatic glands have often undergone caseous degeneration when no tubercles can be found in the lungs; as we have stated, breathing is the commonest mode of infection, and the lymph-glands are the best soil for developing the poison. The course of the disease varies greatly in man; sometimes the general infection immediately follows the local, again years intervene, or it never occurs. The local process cicatrizes or calcifies, and the tuberculous virus becomes innocuous. Many observations prove that tuberculosis is inherited, but we do not know how the semen or ova can carry it; marriages should be avoided among those who are tuberculous or come from tuberculous families. From the great frequency of tuberculosis, and the various opportunities for its inheritance, transfer, etc., why does it not become even more common? *Cohnheim* thinks this is because our generation is acquiring a certain immunity to the virus; he thinks this immunity will also explain why, in some patients, local tuberculous affections do not become general.

Local foci may sometimes be removed by a sharp scoop or by resecting joints, etc., and general infection be thus prevented in some cases; but often the process begins again near the site of the operation, or the granulations covering the wound become tuberculous; cicatrization may still go on but within, the granulations, instead of changing to connective tissue, have tubercles disseminated through them, these become cheesy and suppurate. The common treatment for local tubercle has been removal and cauterization of the surrounding parts with chloride of zinc or iron, caustic potash, etc. Now it is claimed (*Mosetig-Moorhof*) that iodoform has a decided antituberculous action. The wound left after removing diseased parts should be filled with iodoform, then normal granulations develop which change to firm connective tissue, and cure remains perfect. We do not know why iodoform acts thus, but continuous development of iodine takes place on the surface covered with the iodoform; there is no decomposition. Under this application there is little local or constitutional reaction, and only a slight traumatic fever from absorption of non-inflammatory secretion.

The nutrition of patients treated with iodoform almost always improves ; they look well, increase in weight, but in rare cases it has proved a fatal poison ; favorable as has been the effect on the local affections, some patients go on to die of the general disease. *Moleschott* used it internally against tuberculosis, and even claimed that with it he had cured tuberculous meningitis.

Prof. *H. B. Sands, New York Medical Record*, May 25, 1882, gives an interesting article on "Iodoform," in which he states that he has seen two cases of acute mania due to poisoning by external use of this drug. The symptoms were melancholy, delirium, disgust for food, emaciation, profuse sweating. With this as with other substances, fresh wounds absorb more rapidly than granulating ones. The proportion of cases that suffer constitutional effects from the local application of the remedy must be very small.

*Schüller's* experiments led him to believe that anti-bacterial remedies, such as carbolic acid and benzoate of soda, had a directly curative effect on tuberculosis, but this has not been proved.

Hence, the first efforts of treatment should be to increase the resisting powers by good diet, and remove any disease likely to cause tuberculosis ; in the case of limbs, to amputate early ; in cases of catarrhs, to cure speedily. In some cases change of residence to a southern clime for some years may be desirable.]

The new etiology of tuberculosis has given treatment a peculiar and, at a casual glance, a changed position. We now have to ask ourselves the following question: Is there any remedy or mode of treatment by which we can prevent a person, who has on or in him any caseous pus, from being infected with tuberculosis ? To this we must at once say no. The mode of infection is so little known, that on this account alone we could not speak of its prevention. The interval between the development of the primary point of inflammation and the succeeding tuberculous infection is entirely incomputable. In some cases the formation of tubercles in the lungs appears to follow almost on the heels of chronic bronchial catarrh, while in other cases the two forms of disease are separated by years. Typical tubercles may also dry up and become indurated in various ways, or they may rapidly increase, unite, and soften. In short, the variety of the process is very great. But all this gives no starting-point for the treatment. As regards hereditary influence, to which so much importance is properly attached in tuberculosis, some enigmas have been solved by, and some former experiences readily adapt themselves to, the new views. If true tubercle could only develop from infection through the patient himself, of course there could be no talk of direct inheritance of tuberculosis in the strict meaning of the term. Only the tendency to chronic inflammations, ending in suppuration and caseous degeneration, is hereditary; in other words, the scrofulous diathesis, not the tuberculous, is hereditary. We must bear this in mind; the experience of family physicians agrees with it entirely; but we must understand that such general rules are only true in theory. The hereditary tendency to diseases of certain organs, and to certain forms of disease, is such a complicated question that we should be very reserved in stating general laws about it. Apart from the occasional accidental complications, such as meningitis, hæmorrhages, pneumothorax, empyema, peritonitis from perforation of intestines, pyæmia, etc., tuberculosis may prove fatal by extensive suppuration and the rapid febrile marasmus, or by amyloid degeneration of internal organs due to the suppuration, or, lastly, by acute miliary tuberculosis, i. e., by an extensive eruption of tubercles in internal organs, accompanied by general poisoning, where the patient is in a typhoid state. In the earlier stages recovery may take place, but leaving a tendency to relapse.

If we put together what may be said about the indications for treatment of tuberculosis, it would be about as follows: We cannot prevent either the development or progress of tubercles. Hopeless as this sounds, it remains to be added that medical care may accomplish something in hindering the development of those processes which are so often followed by tuberculosis. The early, careful, general dietetic and local treatment of chronic diseases of the bones and joints, and even the amputation of limbs, or the resection of bones at the proper time, may prevent the development of tubercle. In the same way, great care of catarrhs of all sorts, and their most perfect removal, is undoubtedly the most effectual thing we can do to remove the tuberculous infection. In tuberculosis the treatment is the same. All the remedies, baths, and places for treatment, that are prescribed, have for their object—**1**, to remove or diminish the existing catarrh or other primary disease; 2, to improve the nutrition of the patients, who are generally emaciated; 3, to avoid every thing that can render the patients feverish. I must leave it for the lecturer on clinical medicine to make you better acquainted with the important principles of treatment in this frequent and fearful disease.

3. *Arthritis*, or *gout*, is a tendency to disease which usually appears first about the thirtieth to the forty-fifth year of life and later; it is often confounded with chronic rheumatism, but really differs from it considerably. True gout is a rare disease with us, and is distinguished from rheumatism by the fact that it occurs in attacks, often recurs only once a year, or at stated intervals, while meantime

the individual remains perfectly well. Gout is a disease of the rich, and, as old physicians who had it themselves used to say, of wise men. It occurs chiefly in men who lead a comfortable, inactive life; it not unfrequently descends to the next generation, but always appears first after middle age. *Harvey*, *Sydenham*, and many other celebrated physicians, suffered from gout. The inflammations occurring in gout are chiefly limited to certain joints, and the parts around them. The joint between the metatarsus and the first phalanx of the big toe is affected particularly often; this is the seat of true podagra. The wrist and the joints of the phalanges may also be attacked by gout; here it is called chiragra. The skin over the joint is implicated in these inflammations. During the attack it becomes bright red and very sensitive, as in erysipelas; and, in rare cases, ulcers may form during this process. Arterial thickenings (atheroma of the artery), with their occasional results, cerebral apoplexy and senile gangrene, are not unfrequent in arthritic patients. Corpulence, diseases of the liver and kidneys, may also accompany gout; gravel, especially a fine granular excretion of uric or oxalic acid from the kidneys into the bladder, is not unfrequent, but, just as frequently, large renal and vesical calculi develop. In the diseased joints and sheaths of the tendons considerable quantities of urates have been seen, occasionally in such quantities that they covered the articular surfaces and capsule like a white granular coating. An attack of gout is usually preceded for some time by a general feeling of being out of sorts, which disappears as soon as the inflammation attacks some external point, usually a joint. These inflammations last two or three weeks, and then subside, often leaving permanent thickening of the joint; but in other cases the diseased limbs often remain unchanged for years. In some old arthritic patients these stone-like gout-nodules are also found in the skin, as in that of the ear, as well as in the joints and sheaths of the tendons. If these nodules break off, the masses of lime and urates may be scooped out with an ear-spoon; the complete suppuration and closure of these open and very painful gouty nodules then last for months. Operations with the knife in such cases should be carefully avoided. The ordinary attack of podagra never ends in suppuration, always in resolution. — From this etiological relation of the abnormal deposits of uric acid to the joint affection, gout has also been called arthritis urica.

The treatment of the attack of gout, of the gouty articular inflammation, is to be distinguished from the general treatment. The former almost always runs a typical course, which is not materially changed by treatment. The first indication for medical aid is to alleviate the pain by moderating the inflammation; for this purpose ice might answer very well, if there were not certain reasons for fearing its effects, for, from the frequent presence of atheroma of the smaller arteries, great cold might induce gangrene. There is not much to be said against the application of cold compresses, cold fomentations with lead-water, weak solutions of nitrate of silver, or local applications of leeches; but many gouty patients prefer greasing the joint and wrapping it in wadding. Profuse diaphoresis, induced by hot tea and hydropathic packing, is said to shorten the attacks. In the constitutional treatment of the arthritic diathesis, mineral waters take the first rank. Gouty patients should be advised to use the waters of Karlsbad, Kissingen, Homburg, Vichy, and other saline springs, also the thermal waters of Teplitz, Gastein, Wiesbaden, and Aix-la-Chapelle. But we may expect an acute attack of gout to follow the use of warm baths.

4. The *scorbutic dyscrasia* manifests itself in great fragility of the capillary vessels, and consequent subcutaneous hæmorrhages, which result from ruptures of the vessels or from diapedesis, and may be induced in frogs by poisoning them with ordinary salt. This disease is supposed to be due to dissolution of the blood, without any accurate description being given of the blood-change causing the change in the vessels. The disease is almost entirely endemic, for instance, on the shores of the Baltic, and, in a surgical point of view, is not very interesting. When treating of ulcers in the next chapter, we shall refer to it again.

5. The *syphilitic dyscrasia.* Although I do not propose to include syphilis in the subjects of these lectures, still, for the sake of completeness, I must make some remarks on it. This, like the above diathesis, developed in man at some time, but now it is spread entirely by inoculation. The person inoculated is syphilitic from the moment the virus takes effect. In speaking of syphilitic diseases in general terms, three different diseases are included: (1) *gonorrhœa*, a blennorrhœa of the vagina, then of the urethra, which thence occasionally extends to the excretory ducts of the testicles and prostate, and may induce gonorrhœal prostatitis or orchitis; proliferations of the papillary bodies, in form of the so-called condylomata (from κονδῦλος, a button-like prominence on bone), often occur where gonorrhœal pus stagnates; (2) the *soft chancre*, an ulcer, usually on the glans and prepuce, which frequently, through the lymphatic vessels, excites an inflammation of the inguinal glands, which has a great tendency to go on to suppuration; (3) the proper syphilitic ulcer, the *indurated chancre.* In this the general disease occurs at time of inoculation, while the first and second form remain rela local. In inoculation with the secretion of a true syphilitic

the entire organism is infected at once; a series of chronic inflammations occur in the most varied organs, which have at first a more productive character, but soon lead to disintegration of the infiltrated tissue and assume an ulcerative destructive character. The following symptoms may appear in syphilis: eruptions on the skin of blotches, papules, desquamations, and nodules; ulcers in the fauces, on the lips and tongue, and about the anus; osteoplastic and ulcerative periostitis and ostitis, especially on the tibia, cranial bones, sternum, etc.; chronic inflammations of the greatest variety, usually with caseous degeneration in the testicles, liver, brain, and possibly in the lungs. The nodular circumscribed product of syphilis is called by *Virchow* "gummy tumor," by *E. Wagner* "syphiloma." Syphilis may also be inherited; children are born with it; the dyscrasia may be carried by the sperm to the ovum, or be in the ovum. It is still disputed whether a healthy woman who has been impregnated by a healthy man, and has become syphilitic during pregnancy, can convey the disease to the fœtus, and whether a fœtus begotten by a syphilitic man who has no ulcer on the penis can infect the healthy mother. It is also disputed by some that the venereal poison can pass through the placenta.

Gonorrhœa and the soft chancre are local diseases, and are to be treated as such. Formerly soft and indurated chancres were regarded as two forms of syphilis, with many connecting links; of late the dualistic theory seems to gain more and more supporters, although there is still much discussion on the subject. Many surgeons consider mercury as a specific, or as a sort of antidote, in syphilitic dyscrasia. It seems to me proved by recent observations that this is not exactly true. Constitutional syphilis, which only attacks a person once, may in the course of time be to some extent gotten rid of by the change of tissue; hence all remedies that greatly promote the change of tissue are in a certain sense antisyphilitic. Most frequently treatment by sweating or purging is resorted to; occasionally syphilis is cured by a treatment of six weeks; in some cases these modes of treatment must be continued with interruptions till they prove successful, and, finally, some cases are entirely incurable. Occasionally mercury, by inunction or internally, in various preparations, continued a long time, removes the symptoms of syphilis with surprising rapidity; and hence, in cases where we desire to arrest as quickly as possible certain ulcerative forms, especially in the bones, it will maintain its value. Of late it has been much doubted if mercury alone can cure syphilis, and at the same time it has been shown what injury may be induced by continued use of mercurials, by a sort of chronic mercurial poisoning (hydrargyrosis). The mercurialists and anti-mercurialists have disputed for a long time; and in the last decennium it has entered new stages, without, however, having brought all physicians to a conclusion on this question. I incline to the views of the anti-mercurialists. In the course of your studies you will hear still more about this important and interesting point. Iodide of potash is generally recognized as one of the most important and efficacious remedies for syphilitic diseases of the bones and glands, while it does little good in other syphilitic diseases.

[Rapid cures of syphilis are rare ; and many patients, apparently cured, in after-years have syphilitic eruptions or severe forms of syphilis of the brain or other internal organs. Hence, we should guard against being deceived by the apparent effect of a short treatment ; but with proper changes this should continue two or three years.

Of late it is claimed that in the secretions from gonorrhœa, chancroids, and chancres, micrococci characteristic of the different diseases have been found, but this lacks confirmation.]

## LECTURE XXX.

Local Treatment of Chronic Inflammation: Rest, Compression, Resorbents, Antiphlogistics, Derivatives, Fontanels, Setons, Moxæ, the Hot Iron.

It still remains, at the close of the chapter on chronic inflammation, to run through the remedies that we may employ locally, and which are more or less prominent according to the case. Where we do not succeed in finding a constitutional cause for a chronic inflammation, we are limited to local remedies.

*Absolute rest* of the inflamed part is necessary in all cases where there are pain and congestion. When possible, these are combined with elevation of the diseased part, by means of suspensories or pads placed beneath. This, by facilitating the return of the blood, has the effect of relieving and finally removing the venous tension, which is favored by the absolute rest, and hence is especially important in cases where venous congestion has induced or increased chronic inflammation.

*Compression.* This is applied by wrapping the diseased part with moist or elastic bandages, plaster-dressing, strips of adhesive plaster, or even by covering with moderate weights (as in compressing swollen inguinal glands). Compression is one of the most important, and, when made to act regularly, is the most certain means of removing chronic inflammatory infiltrations.

*Massage*, of which we spoke when treating of distortions, is particularly serviceable for getting rid of old infiltrations; it sometimes accomplishes wonders; but this method of treatment must be followed with great energy and perseverance.

*Moist warmth* in the form of cataplasms, continually applied, is also very efficacious, as are also the *hydropathic wraps.* These are applied by dipping a cloth, folded several times, in cold water, wringing it out, enveloping the affected part with it, and covering with some air-tight substance, such as oil-silk, gutta-percha cloth, etc., and renewing this dressing every two or three hours. The skin, at first much cooled, soon becomes very warm; then the dressing should be renewed, so that the cutaneous vessels are kept active by the change from cold to warm, and are thus placed in the best state for absorbing. In some cases these wraps are very useful.

*Resolvent remedies.* Fomentations with lead-water, infusion of arnica, camomile-tea, etc., have some reputation as resolvent applications, which they do not, however, deserve; they rather belong to the category of inactive domestic remedies. Mercurial salve, mercurial plaster, ointment of iodide of potassium, and tincture of iodine, are also absorbents which may be employed alternately in chronic inflammations. I am far from denying them any efficacy in such cases; but you must not expect too much from them. Of late, tincture of iodine, in doses of 5–10 drops, has been injected into lymphatic glands, but with very uneven effect. I pass over a series of resolvent plasters; they do little good in this way; their effect is partly as slight irritants to the skin, partly as protective coverings; in some cases I order such plasters to prevent the patient from applying something injurious; mercurial plaster only has a medicinal effect when used for a long time. I may mention electricity as a discutient remedy; its effect does not seem to be very great, but cases are reported where it has been used with advantage; further investigations should be made on this point.

Antiphlogistic remedies proper, such as ice, leeches, cups, etc., about which you will learn in the clinic, are rarely used, and are only of slight temporary benefit in chronic insidious inflammations; but, in intercurrent acute attacks, they are just as useful as in primarily-acute inflammations. Some surgeons of the present time, especially *Von Esmarch*, use ice continuously in chronic torpid inflammations, and praise the result of this treatment.

*Derivatives.* These play an extensive *rôle* in the treatment of chronic inflammations. They are so named because they are said to remove the inflammation from its location to other points where it will be less dangerous; there are remedies by which we may induce cutaneous inflammations of varied grades, and which have been proved by careful observers to have an excellent curative effect. The physiological explanation of the mode of action of these derivatives is as yet an unsolved problem. It is supposed that, from the application of these remedies near a point of chronic inflammation in a bone or joint, the blood and fluids are drawn outward to the skin. In some cases of inflammation accompanied by little energy or vascularization, the derivatives certainly have rather an opposite effect; i. e., the new acute inflammation induced in the immediate vicinity of the chronic one causes stronger fluxion to these parts, and arouses the chronic, torpid inflammation into an energetic, active state. But we shall not worry ourselves trying to discover the physiological way in which these remedies act; this has always been a very thankless task. The following remedies of this class are practically useful: *Nitrate of silver* in concentrated solutions mixed with fat, and rubbed on the skin a couple of times daily, induces a dark-brown hue, with silvery lustre in the skin, and a slow detachment of epidermis. It is one of the mildest derivatives, and is particularly suited to the joint diseases of sensitive children. *Tincture of iodine*, especially the strong tincture (iodine ʒ j to absolute alcohol ℥ j dissolved with ether), if applied to the skin morning and evening, induces a tolerably sharp burning pain; if this painting be continued two or three days, the epidermis is elevated into a vesicle, occasionally all over the space where the remedy has been applied. *Blistering plasters* act more rapidly; they consist of powdered cantharides (lytta vesicatoria, melŏe vesicatorius) rubbed up with wax or fat, and spread on linen, leather, or oiled muslin. Well-made ordinary emplastrum cantharidum, in pieces as large as a franc or a dollar, is fastened on the skin, and in twenty-four hours a vesicle forms under it; this is to be punctured, and a piece of wadding applied over it; this dries on and becomes detached in three or four days, at which time the detached hard layer of the epidermis has been regenerated from the rete Malpighii. A large spanish-fly blister may be applied once, or a small one may be applied new every day; the latter method is called vesicatoires volantes. Lastly, we may apply plasters containing only a small amount of cantharides, and only inducing continued redness. This is the emplastrum cantharidum perpetuum, or emplastrum euphorbii; it is worn several days or weeks in succession. Although the favorable action of the above derivative remedies in chronic inflammation cannot be denied, I may say that particularly tincture of iodine and blisters do much more good in subacute inflammations, or the slight intercurrent acute attacks in chronic inflammation, than in the painless torpid forms.

The remedies still left to mention are those followed by long-continued suppuration, a suppuration which is kept up by artificial external irritation, according to the will of the physician. Their use is so diminished during the last ten years that at present very few surgeons resort to them.

*Tartar-emetic ointment* and *croton-oil.* When repeatedly applied to the skin for a length of time, in about six or eight days, or in irritable skins earlier, both of these induce a pustular eruption, which is not unfrequently painful. When these pustules begin to show themselves, we stop the applications and allow the pustules to heal. Considerable cicatrices not unfrequently remain; the effect of these remedies is rather uncertain, so that they are not often used.

By fonticulus or a fontanel (from fons, well), we mean an intentionally-induced wound of the skin that is kept suppurating; it may be induced in various ways. You may apply an ordinary blister-plaster, then cut the blister and daily dress the part denuded of epidermis with ointment of cantharides or other irritating salve. You will thus induce a suppuration that you may keep up as long as you continue this mode of dressing. Another way of making a fontanel is to incise the skin and place a number of peas in this incision, retaining them in position by adhesive plaster. The peas swell up, and are to be daily renewed; they irritate the wound as foreign bodies; a simple ulcer is thus artificially induced. It is always simplest to make the fontanel with an incision, but we may burn the skin thoroughly with any caustic, and keep the resulting wound suppurating by the introduction of peas.

The *seton* is a small strip of linen, or an ordinary lamp-wick, which is drawn under the skin by means of a peculiar needle. The seton-needle is a moderately-broad, rather long lancet with a large eye at its lower end, to carry the seton. Setons are generally applied to the back of the neck in the following manner: with the thumb and forefinger of the left hand you lift as large a fold of skin as possible, transfix it at its base with the threaded seton-needle and draw the latter through. After the seton has lain quiet a few days, and suppuration begins, pull it forward and cut off the part impregnated with pus; repeat this daily. Granulations form in the whole canal occupied by the seton; these secrete quantities of pus. The seton is worn for weeks or months, and removed when we wish the suppuration to cease.

Another mode of inducing continued suppuration is by making a slough in the skin by means of heat and preventing the resulting granulating wound from healing by irritating dressings or by introducing peas; this may be kept up a longer or shorter time, according to the effect desired. For this purpose there are two modes of operation, by the so-called *moxa* and by the *hot iron.* Moxæ are thus prepared: a wad of cotton is tied together with silk thread, then soaked in spirits, held on the skin with forceps and there burned. Various grades of burn may be induced by the longer or shorter action. There are other modes of preparing moxæ, which, however, I shall not here describe, as moxæ are now little used. If you wish to induce a slough in the skin, it may be most simply done by strong caustics and *caustic pastes*, or by the hot iron. The cautery-irons used in surgery, already mentioned among the hemostatic remedies, are thin iron rods a foot long, with wooden handles, and with a button-shaped, cylindrical, or prismatic end, which is placed in a basin of hot coals till it reaches a red or white heat. With this, various grades of burns, even to charring the skin, and burns of variable size, form, and depth, may be induced, according as we desire extensive suppuration, or several distinct small ulcers.

It would lead me too far, and not be very comprehensible for you at present, were I here to enter into an exhaustive criticism about the choice and various gradations of the above remedies. These are things that you learn more quickly and certainly in the clinic, from the remarks on an individual case. I will only observe that the application of the more intense derivatives, such as fontanels, moxæ, setons, and the hot iron, to children and susceptible, delicate persons, should be made very carefully, and had better be avoided. I scarcely ever use the hot iron as a derivative, though I sometimes employ it to destroy spongy granulations in caries, occasionally with very good effect.

Almost all classes of remedies have for a time been somewhat the fashion, according to the prevailing theories, and so there was a time when moxæ, the hot iron, or fontanels, were praised as universal remedies in every chronic inflammation. A fontanel was applied on the arm to protect the person against rheumatism, hæmorrhoids, tuberculosis, or cancer, with the idea that with the pus from the fontanel all morbid juices, the materia peccans, were thrown off from the body. In the same way, formerly, at certain seasons, purgatives, emetics, venesections, etc., were resorted to yearly. Even at present you will hear old practitioners tell gleefully how this or that patient was preserved from a multitude of ills by the application of a fontanel. I shall not presume to criticise what may be accomplished by this treatment, for, as was mentioned, we are far from knowing how to measuse its physiological effect; but we should mistrust the action of remedies that are recommended against all possible diseases.

[We may apply a rubber bandage, or (especially for swollen joints) a moistened compressed sponge kept in place by bandage. The compression may even be applied by *Esmarch's* bandage for a short time.

Massage, of which we spoke when treating of sprains, is particularly applicable for getting rid of old infiltrations; it sometimes induces speedy suppuration, and the case goes on as one of acute abscess, or the chronic inflammatory products are rendered fluid and absorbed by the excited blood and lymph circulation. Under it, thickenings dependent on true imflammatory neoplasia may disappear rapidly; even coagulated fibrine, such as we sometimes find in chronic inflammations of the nerve-sheaths, may be reabsorbed. Besides its local effect, massage may stimulate the kidneys to increased secretion, and thus cause absorption of œdema even when its local cause cannot be cured.]

The rapid absorption of old torpid infiltrations is sometimes very favorably affected, as are neuralgic pains in chronically-inflamed parts, by warm or hot local *mud-baths.* In some parts of Hungary hot springs open into the mud of small streams; in this natural hot mud, which is used in tubs, the diseased limbs are soaked once or twice daily; similar baths are prepared artificially. The bog-baths at Franzenbad and Marienbad are about as efficacious; the bog, soaked with ferruginous water, is warmed and used as the mud-baths above described. We do not know whether the mineral salts contained in the bath have any effect; they probably act only as large cataplasms. Compresses wet with thermal water of iodine-springs have also a good reputation as resorbents. Usually they soon induce cutaneous eruptions, and may also be considered as derivative remedies. *Animal-baths* are also very popular; in these the diseased limb is placed among the intestines of an animal just killed, and kept there till the dead body is cold; a peculiar effect is claimed for the animal warmth, of which I have been unable to convince myself. Lastly, we must mention hot *sand-baths,* which were formerly very popular; these probably have no advantage over moist warmth.

# CHAPTER XV

## *ULCERS.*

## LECTURE XXXI.

**Anatomy.**—External Peculiarities of Ulcers; Form and Extent, Base and Secretion, Edges, Parts around.—Local Treatment according to the Local Condition of the Ulcer; Fungous, Callous, Putrid, Phagedenic, Sinuous Ulcers, Etiology, Continued Irritation, Venous Congestion, Dyscrasial Causes.

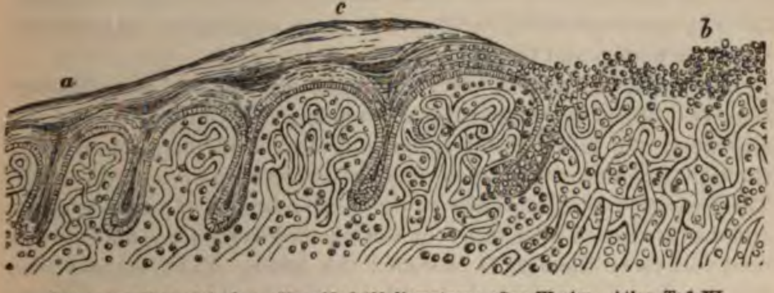
THE study of ulcers naturally follows that of the chronic inflammations. Physicians practically agree as to what an ulcer is, and whether any given wounded surface is to be so regarded; but, to give a short definition of it is about as difficult as it is to define any other object in medicine or natural history. To give you a proximate description of it, we may say, an ulcer is a wounded surface which shows no tendency to heal. Here you see at once, that every large granulating wound with free proliferations, which halts in its progress toward cure, may also be regarded as an ulcer, and, in fact, *Rush*, to whom we owe our most comprehensive nomenclature of ulcers, designates granulating wounds as ulcus simplex.

From personal observations and examinations we conclude that ulceration mostly starts from chronic inflammation, and is always preceded by cellular infiltration of the tissue.

This inflammation may be located in the depth of the cutis, in the cellular tissue, muscles, glands, periosteum, or bones; in the centre of the inflamed spot there is suppuration, caseous degeneration, or some other form of softening and breaking down, with gradual peripheral progression and perforation of the skin from withi outwardly. The *excavated ulcer* is thus formed; as before stated, th is a diminutive cold abscess.

Just as often the process is in the superficial layers of a membran and we have the *open cutaneous ulcer*. We will illustrate this by a example. Let us suppose that from any of the above-mentioned causes we have a chronic inflammation in the skin of the leg, say on the anterior surface of its lower third. The skin is traversed by dilated vessels, hence it is redder than normal, it is swollen, partly from serous, partly from plastic infiltration, and it is sensitive to pressure. Wandering cells are infiltrated, especially in the superficial parts of the cutis; this renders the papillæ longer and more succulent; the development of the cells of the rete Malpighii also becomes more plentiful, its superficial layers do not pass into the normal, horny state; the connective tissue of the papillary layer is softer and becomes partly gelatinous. Now, slight friction at any point suffices to remove the soft, thin, horny layer of the epidermis. This exposes the cell layer of the rete Malpighii; new irritation is set up, and the result is a suppurating surface, whose upper layer consists of wandering cells, the lower of greatly degenerated and enlarged cutaneous papillæ. If at this stage the part be kept at perfect rest, and protected from further irritation, the epidermis would be gradually regenerated, and the still superficial ulcer would cicatrize. But usually the slight superficial wound is too little noticed, it is exposed to new irritations of various kinds; there are suppuration and molecular destruction of the exposed inflamed tissue, then of the papillæ and the result is a loss of substance which gradually grows deeper and wider; the ulcer is fully formed. The accompanying figure is the section of a spreading ulcer of the skin; it formed the basis of this description (Fig. 72).

FIG. 72.



Cutaneous ulcer of the leg. Magnified 100 diameters; after *Förster*. Atlas, Taf. XI.

At *a* you see the cutis already somewhat thickened, toward *b* its papillæ are enlarged, while the vascular loops increase, and the connective tissue is more richly strewn with cells; at *b* is the fully-formed ulcerated surface; at *c* the epidermis is much thickened and forms the indurated border of the ulcer.

On the mucous membrane the process is the same: at first there is a lively emigration of young cells on the surface; this is soon accompanied by a moderate degree of serous and plastic infiltration in the connective tissue of the mucous membrane; the mucous glands secrete plentifully. As already stated, it was believed, until within a short time, that catarrhal pus was of a purely epithelial character; now there is rather an inclination to the view that the elements of catarrhal secretion also are wandering white blood-corpuscles. Continued irritation of a mucous membrane affected with catarrh is followed by softening and breaking down of the tissue, as we described to be the case in the cutis; then we have a *catarrhal ulcer.*

There is another and more acute mode of formation of ulcers, viz.: from pustules that do not heal, but which enlarge after evacuation of the pus, and keep up an acute inflammatory character, as the soft chancrous ulcer. And such ulcers resulting from ecthyma pustules, without any preceptible specific dyscrasia, are particularly frequent on the legs of young, full-blooded, and otherwise healthy persons; we know nothing definite about their causes; they often have a proliferating fungous form, but at other times induce rapid destruction of tissue. But this acute commencement of ulcers is much rarer than the chronic. Some diseases are only half-correctly called ulcers, as the "typhous ulcer;" in typhoid fever there is an acute progressive inflammation of Peyer's plaques, which in many cases ends in gangrene, with necrosis of the inflamed portion of mucous membrane; what remains after throwing off of the slough is a granulating surface, which usually cicatrizes rapidly; strictly speaking, this granulating surface is not an ulcer, it only becomes so when it does not heal normally. Of this, more hereafter; we may use these expressions more freely, when we understand the process perfectly.

From this description you see that, in *ulceration* as in *inflammation*, two opposite processes are combined—new-formation and destruction; the latter results from liquefaction of the tissues, i. e., through suppuration, or molecular disintegration, or both together. There can be no doubt of the antagonistic relations of new formation and destruction to each other in the examples adduced, for it is evident that there the former preceded the latter. But you may also imagine that in a previously healthy portion of skin there might be a disturbance of nutrition of such a nature that disintegration of tissue is the first step, as you have already learned from the section on gangrene. Then on the border of the healthy portion of skin, which retains its vitality, there is a new formation of young cells, and, if the parts adjacent to the primarily necrosed spot be healthy, there must result a granulation surface; but, if the parts be not healthy, and have only a slight amount of vitality, there also we shall have disintegration instead of active inflammatory new formation; an ulcer will thus be formed which will spread gradually. This course, of an ulcer occurring primarily with molecular disintegration without precedent cellular infiltration, rarely presents itself in practice. Strictly speaking, molecular disintegration and gangrene are but quantitative varieties of the same process, viz., the death of certain portions of tissue; cases occur where ulceration and gangrene are very closely associated, as in hospital gangrene, of which we have already spoken; but, as before said, an inflammatory infiltration usually precedes the disintegration.

The above observations, which show the relation of ulceration on the one hand to the new formation, on the other to the gangrene, will have rendered evident the difficulty of preserving systematic divisions of the course of this disease. But do not be afraid that I am going to confuse you: we will enter at once on the special peculiarities of ulcers, you will understand then more readily; here we shall only add that, according to the vital process, all ulcers may be divided into two chief varieties, viz., those where the new formation predominates, which we shall designate briefly as *proliferating ulcers*, and those where suppuration and disintegration are more prominent, which we shall call *atonic* or *torpid ulcers*. Between these two extreme boundary-points of the anatomical and vital peculiarities of ulcers, there are numerous intermediate forms.

To induce healing of an ulcer, the first requirement is arrest of the disintegration on the surface, next that the floor of the ulcer assume, at least approximately, the character of a healthy granulating surface, which goes on to cicatrize in the usual way. In torpid, atonic ulcers it is also absolutely necessary that there should be a free development of vessels and stronger cells, which do not lead to suppuration, but to connective-tissue new formation; in proliferating ulcers, on the other hand, the new formation must be brought back to the normal size. As you will readily perceive, on reflection, this gives the indication for the local treatment to be followed in either case, to which we shall soon refer.

The nomenclature of ulcers varies greatly, according to the peculiarities that are made especially prominent. From the mode of origin, just as in other chronic inflammations, we may distinguish two classes, or chief varieties, viz., idiopathic and symptomatic ulcers. Idiopathic ulcers are such as result from purely local irritation; they may also be termed irritative ulcers. Symptomatic ulcers are such as from some dyscrasia appear as a symptom of constitutional disease, without the action of a local irritation on the affected part. This division of the causes of ulcers is, as already stated, the same that have previously studied in chronic inflammation.

Let us at present leave out of consideration these etiological conditions, and seek first of all, by attending to the external appearances that an ulcer may offer, to give a more perfect representation.

I will only add here that ulceration may not only occur in normal tissue but also in new growths in tumors proper; both excavated and superficial ulcers may form in and on them. In describing an ulcer, the following parts are distinguished:

1. *Form and extent of the ulcer.* It may be circular, crescentic, quite irregular, ring-shaped, superficial, deep; it may be a canal, leading into the deeper parts, a tubular ulcer, a fistula; as I have already told you, these fistulæ result from the formation of a point of inflammation in some deep parts, in a deep layer of the cutis, in the subcutaneous tissue, muscles, periosteum, or bones, or even in the glands, and gradually ulcerating through till it reaches the surface. Hence fistula is always preceded by the formation of an excavated ulcer, of a more or less deeply-seated point of ulceration.

2. *The base and secretion of the ulcer.* The base may be shallow, deep, or projecting; it may be covered with dirty, badly-smelling serous, sanious fluid, or even with gangrenous tags of tissue (sanious ulcers); an amorphous, fatty, creamy, or smeary substance may cover it; it may also have luxuriant granulations with a muco-purulent secretion (fungous ulcers).

3. *The edges of the ulcer* are flat or elevated, wall-like, hard (*callous* ulcers), soft, tortuous (*sinuous* ulcers), zigzag, everted, undermined, etc.

4. *The vicinity of the ulcer* may be normal or inflamed, œdematous, indurated, pigmented, etc.

These universally employed technical terms suffice for the description of any ulcer to a scientific person. But, as the terms expressing the vitality of the process, as torpid, atonic, proliferating, fungous, etc., are briefer, they are more frequently employed; designations referring to the ultimate causes, especially of symptomatic ulcers, are also often used. Thus we speak of scrofulous, tuberculous, syphilitic, etc., ulcers.

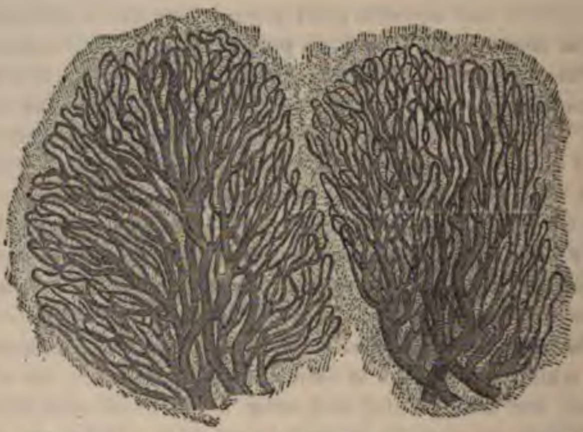
While we have the local conditions of ulcers fresh in our memory, we shall speak of *local remedies*, as far as their employment depends on the condition of the ulcer. A large number of ulcers, especially of those that have resulted from repeated local irritations, heal very readily. As soon as the diseased parts are under favorable external circumstances, and not subject to new irritation, cicatrization often begins spontaneously. It is remarkable how rapidly the common ulcer of the leg begins to improve in appearance as soon as the patient has taken a warm bath, simply applied a wet compress to the ulcer, and remained in bed quietly for twenty-four hours. The ulcer, which previously looked dirty or grayish-green, and had a pestilent odor, looks quite differently; it has a tolerably fair if not very actively granulating surface, and secretes good pus; a fortnight's rest and great cleanliness sometimes suffice for a perfect cure of small ulcers of this kind. But the patient is hardly dismissed, and in his old mode of life, before the cicatrix again opens, and, in a few days, his condition is as bad as ever. So it goes on: the patient again enters the hospital, and is again dismissed, to be again received in a short time We have, however, some means of protection against these relapses, of which we shall speak hereafter. All ulcers are not inclined to heal so quickly; many require various remedies and a long treatment. We shall now run through the various forms, according to their local symptoms, and mention the local remedies to be employed.

1. *The ulcer with inflamed borders, and the erethitic ulcer.* Frequently, while the patient is constantly going about, an ulcer appears very red and painful, and, after a period of rest, this slight amount of inflammation spontaneously subsides. But there are other ulcers whose borders are constantly red and sensitive, the ulcer bleeds easily, and the granulations are painful to the touch. Such ulcers are called *erethitic* or *irritable;* the highest grades of erethism of the surface of the ulcer are very rare; in Zurich, I had a patient, who, as a sequent of a severe inflammation in the thigh, lost a large portion of skin by gangrene; after the detachment of the eschar, there was left a very luxuriantly proliferating, granulating surface, with little tendency to heal, which was so painful to the lightest touch that the patient would cry out and shrink away. The cause of this excessive sensitiveness in such cases has already been mentioned when speaking of nerve cicatrices.

In treating inflamed and erethitic ulcers, we first try mild salves of fresh butter and wax, unguentum cereum, then so-called cooling salves, such as those of zinc and lead, also fomentations with lead-water; if under this treatment the granulations continue painful and look badly, while the inflammation of the surrounding parts is less, we may cauterize the surface of the ulcer freely with nitrate of silver, or, still better, with the hot iron; the latter remedy, with subsequent compression by adhesive plaster, finally caused healing in the case above mentioned. In such cases, the local employment of narcotics is usually recommended, such as cataplasms, with the addition of belladonna, hyoscyamus, opium, etc., but these remedies do so very little good, that, in my opinion, their employment is only time lost.

2. *Fungous ulcers*, i. e., those whose granulations are fungous and proliferating, and project above the level of the skin. These ulcers secrete a muco-pus, and are very vascular.

Fig. 73.



Blood-vessels of two luxuriant granulations of a common (not cancerous) ulcer of the leg, artificially injected by *Thiersch* (Epithelial cancer, Plate XI., Fig. 4).

In these cases we may use astringent remedies and compresses wet with decoction of Peruvian or oak bark, but they are of only moderate benefit. It is best to destroy the surface of such granulations by caustics; daily applications of the solid stick of nitrate of silver usually suffices, where it does not, we may resort to caustic potash or the hot iron. Compression with adhesive plaster is often very efficacious.

3. *Callous ulcers* are most dreaded by surgeons, on account of the long treatment they require; they are those whose base, edges, and vicinity, have become thickened and of cartilaginous hardness, from the long duration of chronic inflammation. The ulcer is torpid, and usually lies deep below the surface; the edges are sharply bounded. The indications for treatment are twofold: first, to soften the tendinous, non-vascular tissue of the hardened borders and base of the ulcer; and to induce a proper amount of vascularity in these parts. We meet ulcers of this variety that have lasted twenty years or more; in such cases we may employ the following treatment: compression, best with strips of adhesive plaster applied in a certain way, as you will see done in the clinic. This dressing, which should cover not only the ulcer but the entire leg, may at first be left on a day or two, but later, when the ulcer begins to heal, it may remain untouched for three or four days, or longer. This so-called

*Baynton* dressing of adhesive plaster is of great service in ulcers of the leg, especially for those cases where the patients are not inclined to lie still, but must attend to their business. In the surgical policlinic of Berlin I made some observations on this treatment of ulcers of the leg, but cannot report so favorably on it, as a *means of cure*, as has been done by other surgeons—they seem to claim that this dressing is an almost universal remedy in ulcers of the leg. I prize it greatly as a protective dressing in dispensary practice, because it enables the patient to go about, without the ulcer spreading; but I cannot see that all ulcers heal particularly well under this dressing, or that the action of the adhesive plaster on the callous borders of the ulcer is more effective than the remedies which I shall mention after a while. The best remedy for keeping up constant congestion in the ulcer, and thus increasing the formation of vessels and cells, is moist warmth, which you may use in the form of cataplasms, or, still better, as a continued warm-water bath. I would particularly recommend the latter to you, for by it you at the same time obtain an artificial swelling and softening of the dry, hardened borders of the ulcer. *Zeis*, who has often employed the warm-water bath in callous ulcers of the leg, also recommends this treatment as one of the most efficacious in such cases. It is sometimes very important to destroy the callous edges entirely, or to excite in them a high degree of purulent inflammation. The former you may most quickly accomplish by the hot iron, the latter by repeated application of tartar-emetic ointment or emplastrum cantharidis. If a pustulous or even gangrenous inflammation of the ulcer and its vicinity be induced by the latter remedies, place the foot in a water-bath and you will often obtain a very quick cure.

It is not always possible to obtain healing of a callous ulcer of the leg; and ulcers along the anterior face of the leg, extending to the periosteum of the tibia, are especially intractable; those ulcers also which surround the leg like a ring are usually reckoned as incurable; they are considered as indications for amputation when they permanently prevent the patient from walking or attending to his business. Besides the above-mentioned circumstances there is still another, that impedes the healing of ulcers with greatly-indurated borders, that is that the healing granulating surface and cicatrix do not diminish and thicken by contraction, because the firmness of the surrounding portions of skin permits no displacement; while, as you know, all granulating wounds decrease to about half their size by contraction, and hence the cicatizing surface grows smaller, in many cases the granulating surface of these ulcers must cicatrize throughout its entire original extent, because it cannot contract. To render this contraction possible, deep incisions have been made through the skin around th ulcer, and these incisions have been kept open by the introduction o charpie; I have never seen any great benefit from this treatmen As a consequence of the rigidity also, the new cicatrix is not suf ciently dense and readily reopens, so that the ulcer once healed so develops again. To guard against this it is best to cover the cicatr with wadding and apply a starch-bandage. This dressing should worn six or eight weeks, till the cicatrix is firm and well organiz I have followed this practice for a long time in all cases of ulcer of leg, and have every reason to be satisfied with it.

4. *Suppurating ulcers.* The causes of decomposition taking place on the surface of an ulcer are often due to unfavorable external circumstances; but, in other cases, from constitutional causes, there is a tendency to more rapid disintegration of the tissue on the surface of the ulcer. Solution of chloride of lime, pyroligneous acid, turpentine, spirits of camphor, and carbolic acid, are the remedies to be applied in such cases. If the destruction of the tissue go on very rapidly, so that the ulcer enlarges greatly from one day to another, it is called an *eating* or *phagedenic* ulcer; this form closely resembles hospital-gangrene above mentioned. In some cases sprinkling powdered red precipitate of mercury quickly arrests the disintegration; should it not do so, I would advise not to postpone the destruction of the entire ulcer; free cauterization with caustic potash or the hot iron, destroying the edges of the ulcer down to the healthy tissue, almost always proves effective in these cases.

5. *Sinuous and fistulous ulcers*—ulcers with excavated edges and fistulæ. They always begin as abscesses, which gradually break through from within outward, and are particularly apt to depend on chronic suppuration of lymphatic glands. Such an ulcer will always heal more rapidly if you make an open ulcer of it, by cutting away the edges of skin, which are usually thin and undermined, or, if they are too thick for you to do this, at least split up the cavity and expose the deeply-seated ulcer. This treatment also answers for fistulous ulcers when they lead to abscesses; the latter must heal before the fistula can close firmly. Let me remark, in parenthesis, the word "fistula" has still another meaning, as it is applied to any tube-like abnormal opening that leads to any cavity of the body; thus w speak of breast, brain, gall-bladder, intestinal, vaginal, urinary, ur thral, and other fistulæ.

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We have still to consider a very important part of the chapter o ulcers, viz., the *etiology*. I have already told you that we have t distinguish local and constitutional causes, just as in chronic inflam mation. Hence all the causes that induce chronic inflammation are again to be enumerated here; we will call particular attention to a few of these. If we first consider more carefully the local causes of ulcers, the most important of them is continued mechanical or chemical local irritation. Continued friction and irritation are frequent causes of such irritable ulcers; a tight boot, the hard edge of a shoe, may induce ulcers on the feet; a rough tooth or a sharp piece of tartar may cause ulcers of the mucous membrane of the mouth or tongue, etc. Ulcers of this variety usually bear the marks of irritation; the vicinity is red and painful, as is the ulcer itself. Among the chemical irritants we have the action of schnaps and rum on the gastric mucous membrane; as a rule, topers have constant gastric catarrh, during whose course catarrhal and specific ulcers, of various kinds, not unfrequently form. A second and still more frequent cause of chronic inflammation, resulting in ulceration, is congestion, especially venous congestion, distention of the veins, varicose veins. These are very intimately connected with the origin of ulcers of the leg; we shall speak of them later (Chapter XIX). There we will only mention that, as a result of the continued distention of the small cutaneous veins, there is chronic serous infiltration of the skin, to which is gradually added cellular infiltration, thickening; and, lastly, there are frequently suppuration and disintegration.

Ulcers due to varices, which are generally briefly termed *varicose ulcers*, may have very varied characteristics. At first they are ordinarily simple, often proliferating ulcers; subsequently they assume a more torpid character, and then the borders become callous. We have already noticed how quickly such ulcers change when they are only treated by rest and cleanliness. In regard to treatment, the already-lauded dressings with adhesive plaster are excellent both for inducing healing of the ulcer and arresting further development of the varices. But in most cases I prefer *rest in bed*, on the principles above given, and only subsequently apply the adhesive plaster to prevent further increase of the varices.

Although we have here shown the intimate relations between varicose veins and ulcers, and have thus called attention to the point of greatest practical importance about this disease of the veins, you must not conclude that varices are always followed by ulceration; on the contrary, there are many cases of enormous varices that are not ollowed by secondary ulcers.

We come now to a short description of those ulcers that are due o internal causes, and are connected with various dyscrasia—the *ymptomatic ulcers.*

1. First among these are *scrofulous ulcers;* these most frequently come in the neck, enclosed collections of pus developing in the cutis or subcutaneous tissue, and gradually perforating out through the skin. Of course, this causes small losses of skin, whose edges are usually red and very thin, and which lead to deeply-seated cavities that evacuate thin pus or tissue that has undergone caseous degeneration. The borders of these cutaneous ulcers are excavated, as may readily be shown by examining with the probe. As a rule, these typical atonic ulcers. From this description you see that this form of undermined sinuous ulcers is only due to the mode of origin, and may occasionally present itself under the most varied constitutional conditions; although experience teaches that it is especially frequent in scrofulous persons, and this is why such atonic ulcers with undermined edges are referred to scrofula. This conclusion will generally prove correct, though it is not necessarily the case.

2. *Lupous ulcers.* By lupus we understand a disease which manifests itself by the development of small nodules in the superficial layer of the skin. The subsequent progress of these nodules may vary. They consist of collections of wandering cells and coincident ectasia of the vessels. Lupous nodules may (*a*) enlarge and run together, so as to form larger nodules and tuberculous thickenings of the skin (*Lupus hypertrophicus*); (*b*) on their surface there is a free exfoliation of epidermis (*Lupus exfoliatus*); (*c*) the surface ulcerates (*Lupus exulcerans*). All three forms may combine, and some others may be added to them. The ulcers resulting from the latter form may be accompanied by strongly proliferating granulations (*Lupus exulcerans fungosus*), or dispose to a more rapid destruction of tissue (*Lupus exedens, vorax*). The disease is most frequent on the face, especially on the nose, cheeks, and lips; it causes the most frightful disfigurement. The nose or the lips may be entirely destroyed by lupus. I saw one case where all the skin of the face, nose, lips, and eyelids, was destroyed; both eyes had been lost by suppuration, and the facial part of the skull, being exposed, presented a most horrible sight. *Dieffenbach* describes such a case in a Polish count, and compares his appearance to that of a death's head. Lupous ulcers do not by any means always look alike; but their surroundings, and the general appearance of the portion of skin diseased, greatly facilitate the diagnosis. When lupus occurs in other parts of the body, as in the extremities or mucous membranes, as the throat or conjunctiva, the diagnosis is difficult, and cannot always be made positively. It is not only pardonable, but sometimes unavoidable, to mistake the disease on the extremities for certain forms of leprosy, and in the throat for syphilitic ulcers. In most cases lupus is due to a dyscrasia. It is

rarely a purely local skin-disease. It is doubtful whether we are justified in claiming a particular lupous dyscrasia, for lupus very often attacks scrofulous persons, so that it may be regarded as one, and one of the worst symptoms of scrofula. It also comes as one symptom of syphilis, so that lupus syphiliticus and lupus scrofulosus are spoken of. Lupus is most frequent during puberty, and attacks females oftener than males; it more rarely develops late in life; beyond the fortieth year we are pretty safe from it.

In the way of *treatment* I attach most importance to local treatment, especially in the ulcerative form, for here we must make every attempt to arrest the progress of destruction, which may endanger all the skin of the face, and internal remedies act very slowly. Here, as in all rapidly-spreading ulcerations, we should radically destroy the base and edges of the ulcer by cauterizing down to the healthy tissue. We generally employ the potential cautery and the solid stick of nitrate of silver or caustic potash, pushing them through the lupus into the healthy parts below. We may also use the caustic in the form of paste, such as chloride-of-zinc paste, which is most readily made by mixing chloride of zinc with rye or wheat flour, and making it into paste with a few drops of water, then spreading it on the ulcer. To attain our object more rapidly, and let the caustic act more intensely, it is advisable to scratch up the floor of the ulcer with the flat end of a probe, and, after arresting the bleeding, apply the caustic. Of the remedies above mentioned, I prefer caustic potash, as it unites with the tissues most rapidly, and consequently the pain ceases sooner. This cauterization may be done during anæsthesia, so that when the patient awakes there will be a moderate and tolerable burning. Nitrate of silver causes the most protracted suffering, but has the advantage of liquefying less rapidly than caustic potash, and hence possesses special advantages for cauterizing some portions of the body. When the slough from the cauterization is detached, if the operation was thoroughly done, there is left a good granulating surface, which cicatrizes in the ordinary manner. A new lupus is not apt to form in this cicatrix, although cauterization cannot prevent the development of new nodules in the vicinity. Painting with tincture of iodine is the best local remedy in exfoliative and hypertrophic lupus. It is well to mix this remedy with glycerine, to render its action less intense. I have repeatedly seen lupus nodules shrivel up under this treatment, but it does not prevent relapses. Lastly, in some cases, the portion of lupous skin may be excised with advantage. The only internal remedy from which I have seen benefit is cod-liver oil, of which four to six table-spoonfuls are to be given daily, but this treatment must be continued for years. Decoctions of barks are only useful in lupus syphiliticus. Arsenic, which is highly prized in other chronic skin-diseases, is of little use in lupus. In Switzerland the disease was rare. My experience of it was chiefly derived in the Berlin clinic, and, if I were to state my belief regarding the efficacy of internal treatment, it would be to the effect that the lupous dyscrasia, like the scrofulous, often disappears spontaneously in the course of time, but is also often incurable.

3. *Scorbutic ulcers.* Scorbutus, or scurvy, is a disease which, as already stated, when fully developed, manifests itself by great weakness of the capillary vessels. There are extravasations of blood at many places in the skin and muscles; the gums swell, become bluish red, and ulcers, which bleed readily, form on them; there are also intestinal hæmorrhages, general emaciation and debility, and many patients die in a miserable state. This severe form of scorbutus occurs chiefly endemically on the coasts of the Baltic, and in sailors on long voyages. In the latter case the disease is usually referred to continued use of salt meat. Inland there is a sort of acute scorbutus, comprising morbus maculosus, purpura, etc. Scorbutus localized on the gums and oral mucous membrane is everywhere common among children; the gums swell, become of a dark bluish red, bleed on the least touch, and ulcers, covered with a yellow, smeary coating of pus, fungi, and shreds of tissue, form on them. When the disease appears in this form, and is treated early, it is generally readily cured. You should paint the gums twice daily with a mixture of half a drachm to one drachm of muriatic acid and an ounce of honey; internally administer mineral acids in dose and form suited to the age, and order a light, easily-digested diet. If this treatment be conscientiously followed, the disease soon disappears. General endemic scorbutus is difficult to cure, because it is generally impossible to withdraw the patients from the injurious endemic influences. In this also the acid treatment is greatly recommended.

4. *Syphilitic ulcers.* The marks that are usually given, as particularly characteristic of syphilitic ulcers, refer almost exclusively to the primary chancre, especially the soft chancre. This begins as a vesicle or pustule, develops to an ulcer as large as a pea, with red borders and a yellow, fatty-looking base. The ulcer of the indurated chancre looks differently; in this there is first a nodule in the membrane of the glans or prepuce. This nodule ulcerates from the surface, as other cutaneous ulcers do. It usually assumes an atonic, torpid character, frequently with a marked tendency to breaking down of the tissue. Broad condylomata, one of the milder evidences of constitutional syphilis, are, strictly speaking, nothing but small, superficial, very circumscribed fungous cutaneous ulcers, which occur

most frequently on the perinæum, about the anus, and on the tongue. The so-called *tertiary syphilitic ulcers* of the skin often have very indurated, brownish-red borders, are circular, or horseshoe-shaped, and are also atonic in character. You will see from this that the appearance of syphilitic ulcers also may vary greatly, and hence that the mere appearance of the ulcer does not enable us to judge with certainty of the presence of constitutional syphilis. The treatment of true syphilitic ulcers should be chiefly internal, and be directed against the constitutional disease. Locally we should use intense caustics if the destruction of tissue is going on rapidly.

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Older surgeons also distinguished numerous forms of ulcers that have not been mentioned here, and that were said to be characteristic of the causes. For instance, in his treatise on ulcers (Helkologie) *Rust* speaks of rheumatic, arthritic, hæmorrhoidal, menstrual, abdominal, herpetic, etc., ulcers. But I, in common with other surgeons of modern times, have been unable to penetrate into the mysteries of this exact diagnosis. It is now generally considered that the old nomenclature was based rather on an artificial system originating in the old humoral pathology than on critically exact observation. From unprejudiced observation we should unquestionably acknowledge that certain forms of ulcers, particularly when affecting certain localities, enable us to decide on their cause; nevertheless, the appearance and form of the ulcer are very dependent on the anatomical relations of the part affected (e. g., as by the course of the filaments in the skin, *Wertheim*), and on various external causes, so that we should frequently be deceived if we relied too much on the appearance of the ulcer as an unmistakable expression of a specific constitutional cause.

[A mode of treating ulcers of the leg, uniting moist warmth and compression, is by the so-called *Martin's* bandage of thin rubber, which is applied directly over the carefully-cleansed ulcerated surface merely tight enough to compress but not to be painful. This may be put on in the morning before rising, the patients being allowed to go around under this dressing; the callous infiltration gradually softens, and perhaps contact with the rubber may have some good influence. At night the bandage is removed, and some simple salve or moist warmth applied. Massage is another remedy, not yet much used, which has proved useful where there was hard, callous, immovable skin about old ulcers; but this must be tried very persistently.

In some cases where the ulcer cannot be cured, constantly wearing a rubber bandage, or elastic stocking, enables the patient to work and be comfortable. As the results of amputation become more favorable, it is very likely that chronic ulcers of the leg will more frequently be regarded as indications for amputation than has been the case.

In *lupous* ulcers, simple cauterization is far surpassed by scraping the suspicious spots; the diseased part comes away readily, while the healthy tissue resists the scoop; after scraping, the base should be cauterized.

Another operative treatment for lupus is by making small incisions through the infiltrated skin with a lance-shaped knife; the bleeding is considerable; when it has been arrested, apply moist compresses. Sometimes this requires repetition at intervals. It is said that the resulting cicatrices are less perceptible than those from scraping and cauterization.]

# CHAPTER XVI.

## *CHRONIC INFLAMMATION OF THE PERIOSTEUM, OF THE BONE, AND NECROSIS.*

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## LECTURE XXXII.

Chronic Periostitis and Caries Superficialis.—Symptoms.—Osteophytes.—Osteoplastic, Suppurative Forms.—Anatomy of Caries.—Etiology.—Diagnosis.—Combination of Various Forms.

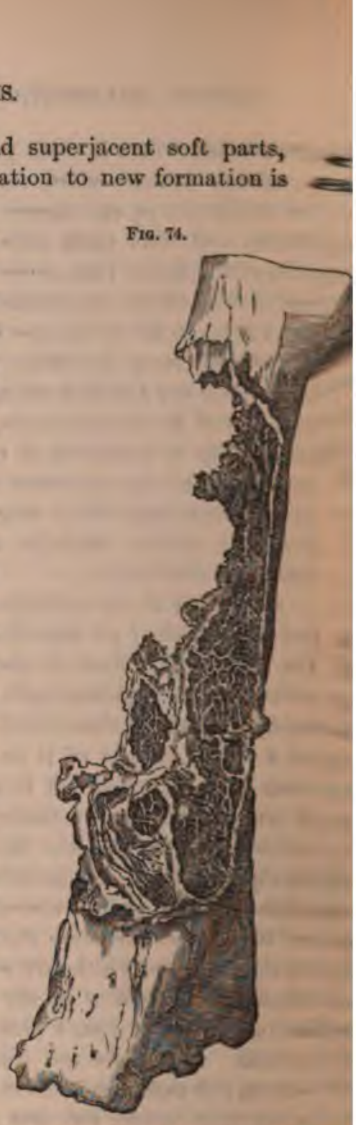
GENTLEMEN: Chronic inflammations of the bones and periosteum, to which we now pass, are far more frequent than the acute forms; the more common disease is chronic periostitis, which is often accompanied by ostitis (caries) superficialis. In the early stages this may end in resolution, then go on to suppuration, with ulceration of the surface of the bone; it may also be accompanied by a deposit of newly-formed ossific substance on the surface of the bone. Periostitis that has lasted some time will never leave the bone unaffected. Let us first consider the *symptoms of chronic periostitis.* The first symptoms are usually slight pain, and moderate swelling of the parts immediately around the affected bone. These are accompanied by slight functional disturbances, especially when the disease is in one of the extremities. Spontaneous pain is usually slight, or may even be entirely wanting. Pressure induces severe pain, and we find that the impress of the finger remains evident on the skin for some time, showing that the swelling of the skin is chiefly œdematous. The disease may remain for a long time in this stage, and may subside as gradually as it began. In such cases you may consider the affection as located in the external loose connective tissue of the periosteum. Here there is distention of the vessels, serous and plastic infiltration.

The symptoms above given may also depend on a periostitis combined with a *superficial* ostitis, only in the latter case the spontaneous pains are occasionally more intense; there are also severe, boring, tearing pains at night. If such a process has lasted for months a [illegible] then recedes, the affected bone remains thickened and nodular on [illegible] surface. If you have a chance to examine such a case anatomica[illegible] you find the following: The two layers of the periosteum cannot [illegible] exactly separated; both have changed to a fatty-looking, tolera[illegible] consistent mass. On microscopical examination you find that the [illegible] sue consists of connective tissue richly strewn with cells and trave[illegible] by dilated capillaries in greater or less number. This morbidly-th[illegible] ened periosteum is more readily detached from the surface of the [illegible] than is normally the case; the subjacent bone (we are supposing a hollow bone, such as the tibia) has its surface covered with small nodules of peculiar, occasionally stalactite shape. If you now saw through the bone, you find that these nodules on the still-distinct surface of the compact cortical substance are a thick layer of porous, apparently young, newly-formed bone-substance, which are very intimately connected with the cortical substance, it is true, but which, nevertheless, if the process be not too old, may be broken off with a chisel in good-sized pieces. If the disease has already lasted some time, and the union has become very intimate, we find that the deposited porous bone has become more compact, especially if the morbid process has actually terminated.

Let us stop here a moment to inquire the origin of this newly-formed bone. It may come either from the inner surface of the periosteum, or from the surface of the bone. The former is the generally-received opinion, and it is supposed to be a renewal of the function of the periosteum, as it existed before the bone had completed its growth, when regular layers of new bone were always formed on the inner surface of the periosteum. This form of periostitis, which is combined with the formation of *osteophytes* (as the young bony substance deposited during inflammation is termed), may be called *osteoplastic*, a name which I shall use, for the sake of brevity. Nevertheless, I do not agree in the above view, that osteophytes proceed solely from the periosteum, but am satisfied that they actually grow from the bone, as the Greek name indicates. For, microscopic examination shows that, in this case also, as in suppuration and granulation on the surface of the bone, the small vessels that enter and escape from the bone with their enveloping connective tissue are the seat of the new formation, which advances from the Haversian canals opening on the surface of the bone, and are the point of origin for the new formation of bone, which then spreads out under the periosteum. These ossifying granulation-nodules grow from within outward somewhat into the periosteum, and then the latter takes secondary part in the process, as it seems to me. The form of [illegible] osteophytes, which is often peculiar, depends on the arrangement of the vessels around which the young osseous material is deposited. We would not by any means assail the undoubted fact that the periosteum, and other parts adjacent to the bone, may also produce new bone, still I assert that, correctly viewed, osteoplastic periostitis is an osteoplastic ostitis superficialis. This subtle distinction has no practical value, so far as we now know. *Osteophytes are the product of an inflammatory irritation of the periosteum and surface of the bone; they are precisely what we call callus, in fractures, and they are formed in the same way.* I here remark that periostitis, accompanied *only* by formation of osteophytes, without any suppuration, is especially peculiar to some forms of constitutional syphilis. The dolores osteocopi, which may be so torturing in the head and shinbones, in tertiary syphilis, are almost always due to osteoplastic periostitis and ostitis.

According to my experience, almost every chronic periostitis is at first osteoplastic; all other terminations follow it more or less closely. The *suppurative* form is also very frequent; it may run its course without the bone being much affected. Recall the symptoms already mentioned: œdematous swelling of the skin, pain on deep pressure, and a slight amount of it on moving the limb. This condition remains long the same, but is gradually followed by more swelling, by an immovable, doughy tumor, not perfectly but still tolerably well defined. By degrees the skin reddens, and the tumor fluctuates decidedly. Four to six months may thus pass, and then the tumor remains for a long time unchanged. The pain has probably increased, and the function is more disturbed. If the disease be left to itself, the cold abscess, which now evidently exists, will open, and a thin pus mixed with flocculi or cheesy substance will escape. If, through the fine opening, you pass a probe, it will enter a cavity lined with granulations. If you do not wait for the spontaneous opening of the abscess, but make an incision through the thin skin, it is possible that no pus may escape, but that you will find the fluctuating tumor to consist of a gelatinous mass of red granulations; in other cases there is some pus in the centre of the swelling; in still others the entire tumor is of pus. From what I have already told you of the anatomical conditions in chronic inflammation, you will readily understand these different states. If, in the periosteum, infiltrated with serum and plasm, you imagine a rich development of vessels, and at the same time an infiltration of wandering cells, and transformation of the connective tissue to a gelatinous intercellular substance, the former is metamorphosed to a spongy mass of granulations. This may sooner or later change to pus, and an abscess is the final result. If the whole process affects only the periosteum and superjacent soft parts, bone is but little changed; some inclination to new formation is hibited on its surface by the production of a layer of osteophytes under and in the periphery of the part affected with periostitis. Nevertheless, there is a possibility of the abscess healing slowly, after the pus has been evacuated, and of a return to the previous normal state. Such a recovery of periostitis, without implication of the bone, occasionally occurs in practice, but it is rare. It is far more common for the bone to be also affected, perhaps only superficially; that is, for periostitis to be accompanied by ostitis; not an ossifying, but a chronic, suppurative, ulcerative ostitis — a *caries superficialis*. Before the abscess has opened, the symptoms of such a caries scarcely differ from those of suppurative periostitis. If the abscess has opened, we may pass a probe into the surface of the bone, which we feel to be rough and gnawed. The caries had existed some time, and was secretly eating into the bone before the abscess opened; it probably existed when the periosteum only appeared infiltrated, and was still in the stage of gelatinous granulation. Hence, suppuration is not necessarily combined with caries, although it frequently accompanies it. To make all this clear to us, we must study chronic ostitis by means of preparations. The whole development and course are quite analogous the course of chronic inflammation in the soft parts, but the hardne and difficult solubility of bone give rise to somewhat different circu stances.

FIG. 74.



Caries superficialis of the tibia, acco ing to *Follin*.

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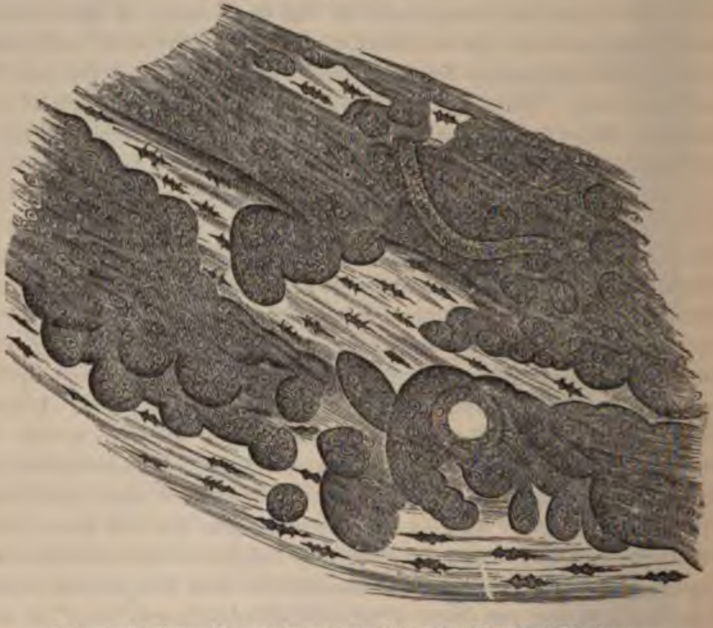
In the course of these lectures we have repeated time and ti again that inflammatory neoplasia is developed in and from t affected tissue; that the close connective-tissue filaments, by rich filtration of cells, are transformed into gelatinous or even fluid intercellular substance. Now, how shall this be transformed into bone? The cells embedded in the stellate bone-corpuscles participate no more in the inflammatory new formation than the stable connective-tissue corpuscles. Here also, as in most tissues of the body, the inflammatory neoplasia infiltrates the connective tissue; namely, that which envelops the vessels in the Haversian canals, and in the medulla of the bone. Still, the space for the extensive production of cells is limited, and, if the wandering of the cells went on very rapidly, the vessel would soon be entirely compressed in the bony canal; if the circulation be then arrested, the nutrition of the young brood of cells also ceases, and the necessary result is death of the affected portion of bone (necrosis). Quite right, this may be the course; superficial necrosis may thus combine with periostitis; of this hereafter. Usually, however, the cell infiltration in the Haversian canals is not so rapid as to compress the vessels. The process is chronic; the bone gradually gives way, the Haversian canals become wider and wider, the firm cortical substance of the bone becomes porous, in the canals (widened to meshes) lies the brood of young cells, interspersed with gelatinous intercellular substance and numerous vessels, an *interstitial proliferation of granulations.* If you imagine the process as continuing, the bone disappears more and more, the entire infiltrated portion may be dissolved, and the inflammatory neoplasm takes its place. If you macerate such a bone, at the seat of disease you will find a loss of substance, with rough porous walls, that look as if gnawed off; in this defect lies the neoplasia that has taken the place of the bone (Fig. 74). Now, remember that so far the word pus has not been mentioned; still, of course, the inflammatory neoplasia may subsequently suppurate, and, if we continue our supposition that the process began in the periosteum, you have a superficial cold abscess lying on the bone; its walls may be covered with granulations.

If you have carefully followed me thus far, you will have remarked already that throughout the whole process the bone substance remains entirely passive; it is entirely consumed, and we might say, with a certain amount of truth, chronic ostitis, or caries, is actually only a chronic inflammation of the connective tissue in the bone, with consumption of the latter. And according to my view this is perfectly correct, at least for the great majority of cases. Still, how does this consumption of bone take place? Should not microscopical examination show whether the bone-cells are changed or not during the process? Remove with the forceps a particle of bone, as thin a sheet as possible, from a carious spot, and look at it under the microscope, you will in many cases see its edges and surface bitten out, as it were;

the bone-corpuscles are unchanged; the intercellular substance somewhat more cloudy than usual, perhaps, but not much altered; a section of bone, taken from the vicinity of such a carious spot, shows nothing different. If you saw or cut out a piece from a carious spot, and abstract the chalky salts from the bone by chromic acid, and then make sections through it and clear them with glycerine, you will have about the following picture (Fig. 75);

FIG. 75.



Section of a piece of carious bone (caries fungosa). Magnified 350 diameters.

These pieces of bone are often bitten out, as it were, quite regularly along their edges, the young neoplasia grows into these defects, their further increase goes hand in hand with the dissolution of the bone; the bone-corpuscles are unchanged, no destruction starts from them, we occasionally see them half destroyed at the edge of a piece of the bone. What becomes of the cells that were in them, we can hardly say; they can no longer be recognized among the numerous young cells of the inflammatory new formation among which they enter; it is possible that, freed from their cage, they aid in increasing the cell-brood by subdividing, possibly they die; at all events, as far as may be judged by the change of form, they do not aid in dissolving the bone. But how the bone is dissolved remains an unsolved riddle. Living, like dead bone, may, to a certain extent, be dissolved by the interstitial bony granulation. Previously, when speaking of operating for pseudarthrosis by the insertion of ivory pegs, I told you, if you will remember (p. 244), that the ivory pegs became rough on their surface, carious; there the process is just the same, and this observation is exceedingly interesting and important as a proof that the bone itself does not necessarily have any thing to do with its solution in caries, but may play a perfectly passive part. To anticipate the charge that I admit *only* this variety of consumption of bone, where the above changes occur on the surface, I must add that I have already called attention to the fact that the ivory pegs introduced for pseudarthrosis do not *always* become rough on the surface, but might remain smooth and still lose substance, as may be shown by weighing them before and after the operation. The morphological appearances in the carious bone, which *R. Volkmann* very aptly designates *lacunar corrosions*, and which *Howship* first made known, are now generally recognized as correct, although different views were formerly held regarding them, which you may find in the cellular pathology of *Virchow*, and in *Förster's* atlas, if the subject interests you.

One point, however, we must consider. It would be very supposable that the bone-substance, having its nutrition affected, would begin to break up and crumble into very fine particles, or powder; this would be especially apt to occur if the bone had previously lost its organic substance. It could even be shown that this is the primary step in ulceration of the bone, or caries, and those who regard destruction of tissue as the primary step in ulcers of the soft parts, and inflammatory new formation as the second, will also hold this view in regard to bone. As I have already stated, my observations speak very decidedly against the universality of this view of ulceration, and what I did not find proven as regards the soft parts, I cannot consider true as regards the bones. But there is no doubt that portions of bone may crumble off, and, when there is suppurative ostitis, these small particles of bone may be found in the pus. This would be a necrosis of the lowest form; such a death of the particles of tissue also occurs in the soft parts, both in acute and chronic inflammation; you will doubtless bear in mind that we have spoken of this subject. It cannot be considered as a rule in caries; it is only seen occasionally in caries with suppuration or caseous degeneration. Here even large portions of bone may become actually necrosed, and for this combination of caries and necrosis we have the curious name of *caries necrotica.*

Thus far we have used the term *caries* as exactly synonymous with chronic ostitis and solution of bone, and at present this is very generally done; but formerly the name caries was only used for ulceration accompanied by suppuration, for open ulcers of the bones. The intimate connection between chronic inflammation and ulceration, which we previously studied in the soft parts, also exists between chronic ostitis and caries. If you desire to designate the character of the inflammation more specifically, it may be done conveniently by certain additions which you already know from the chapter on ulcers. Perhaps it would be better to gradually drop the name caries and replace it by ostitis with various additions, such as rarefying, osteoplastic, ulcerating, granular, etc., or only to employ caries for bony defects caused by lacunar erosions. On macerated bones this is always readily recognized; there we are never in doubt as to whether the bone is carious, for we call carious all defects that look as if gnawed out; they might very well be termed lacunar or corrosive defects. But on living patients it requires accurate knowledge and rich experience to decide certainly whether a bone which a sound enters readily is only softened or has large lacunar defects. Up to this point we have only studied superficial caries; hereafter we shall come to central caries, which holds the same relation to the superficial that the abscess does to an open ulcer. In the soft parts I showed you the development of the process of ulceration in a fungous ulcer, where the productive character predominates. This has its analogy in bone, in *ostitis fungosa* (by caries sicca, *Virchow* and *Volkmann* mean caries with proliferating granulations and destruction of bone without suppuration), where there is as yet no destruction of the inflammatory new formation, but where interstitial granulation-tissue has grown all through the bone. This does not by any means always occur to the extent we have just supposed. If you bear in mind the atonic, torpid ulcer of the soft parts, and remember how the neoplasia rapidly breaks down into pus, undergoes caseous transformation, or disintegrates, and simply apply the same changes to bone, you will readily understand the case; this also gives caries another character; there are very torpid, atonic forms of caries where the neoplasia causes but little destruction of bone, and then disintegrates or undergoes caseous metamorphosis, and thus in the living organism there is a sort of maceration of the diseased bone; the soft parts in the bone suppurate; if this happen *before* the bone is dissolved, the portion of bone that has suppurated is necrosed. Here, also, most of the fault of the disintegration is due to deficient vascularity. But we must look to constitutional influences for the causes why we have in one case fungous or proliferating, in another atonic caries.

We shall become acquainted with other forms of ostitis when we come to speak of primary chronic inflammation in bones.

Chronic inflammation of the periosteum and bone is chiefly due to constitutional, dyscrasial diseases; and although injuries, blows, falls, etc., may be exciting causes of these diseases, the ultimate cause must lie in the injured part or the system at large, otherwise the process would take the course usual to traumatic inflammations and soon terminate. If an injury induces insidious chronic inflammation, this must be due either to a peculiar local or constitutional condition; so far I have had no reason to abandon this opinion.

Of the dyscrasiæ already known to you, the scrofulous and syphilitic especially predispose to chronic periostitis and ostitis; among scrofulous children the fungous forms of caries are most frequent, while among adults the atonic occurs oftener. True tubercles are also found in bone, but, so far as I know, not in the periosteum or the cortical layer of the long bones.

But chronic periostitis also occurs frequently when none of the above dyscrasiæ are discoverable, and where we can recognize no cause; in old people especially, periostitis with caries sometimes comes from very slight injuries, and runs its course in the most disagreeable torpid form.

The inflammatory neoplasia in the bone will greatly sympathize if the general health fails; in children who have died of caries, you will almost always find the atonic form, for, just previous to death, while the nutrition was bad, the neoplasia also broke down; the diseased bone, even during life, was macerated by suppuration and mortification. Pathological anatomists, who only see caries on the dissecting-table, rarely know the fungous form accurately, or consider it the more rare; but, when one often examines pieces of carious bone, cut out during life, especially the resected joints of children, where the process is going on actively, he learns to judge differently from what he would in the anatomical museums, where macerated bones, almost exclusively, are preserved.

Although I have merely spoken of fungous and atonic caries, you still understand that I have only depicted the extremes of the proliferating and rapidly disintegrating new formation. Of course, there are many intermediate forms.

It is not the object of these lectures to carefully delineate all the shades of this process, as will be done in the clinic, but here the picture of diseases should be drawn from typical cases, you should acquire a mental mastery of the subject; hence I only lead you so far into the details of the process as is necessary for understanding its anatomy.

Now you will very justly ask, How shall we know whether the case, which we have only diagnosed with the probe, be of the proliferating or torpid variety? This will have an influence on the treatment, as it has in case of ulcers of the soft parts. And it is important not only for the treatment, but for the prognosis; for pure torpid caries offers far poorer chances than the fungous form, because it is far more apt to occur in poor, badly-nourished, and old persons. The distinction is not difficult. In the more proliferating forms, the swelling of the soft parts, periosteum, and skin, and especially of the articular capsule when the caries affects the articular ends of the bone, is often considerable; all these parts feel spongy. If there be any openings in the skin, proliferating granulations project from them, and a mucous, tough, synovia-like pus escapes. If you examine with the probe, you do not come at once on bare bone, but must push the probe into the granulations, often to some depth, before entering the rotten bone.

In the pure atonic form there is less swelling, the skin is thin, red, and often undermined. The edges of the opening are sharp, as if cut out with a punch; there is a discharge of thin, serous, sometimes badly-smelling or sanious pus; if you introduce the probe, you come at once on the bare, rough bone, from which the soft parts have already been separated by suppuration and maceration. These are the extreme cases of the series; there are various intermediate forms.

Taking all things into consideration, I think you will now have a correct idea of caries superficialis.

Let us make a short review of what we know of chronic diseases of the periosteum and bone. We have considered chronic osteoplastic periostitis (with formation of osteophytes without suppuration), suppurative periostitis alone, and combined with ostitis superficialis, or caries. But osteoplastic periostitis may combine with caries, and this combination is even frequent, i. e., osteophytes form round a carious point in the bone. If you examine a series of preparations of carious joints, you find the osteophytes starting from the surface of the bone, around the destroyed portion; the periostitis, which at one place induced destruction of the bone, caused formation of new bone in the vicinity. You may very aptly compare this to an ulcer with callous edges—thickening by new formation in the periphery, destruction in the centre. But we do not have formation of osteophytes at the periphery in atonic forms of caries; it only occurs in those which, at least for a time, bore a proliferating character; just as in torpid, scrofulous ulcers you only find thickened edges where the skin had for a long time been thickened by plastic infiltration, so in the bone also we have this combination of proliferation and destruction which we have so often met in the study of inflammation.

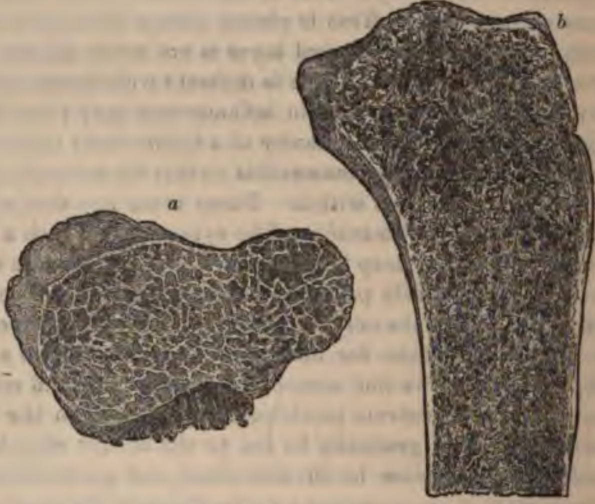
## LECTURE XXXIII.

Primary Central, Chronic Ostitis, or Caries.—Symptoms.—Ostitis Interna Osteoplastica, Suppurativa, Fungosa.—Abscess of Bone.—Combinations.—Ostitis with Caseous Metamorphosis.—Tubercles of Bone.—Diagnosis of Caries.—Dislocation of the Bones after their Partial Destruction.—Congestion Abscesses.—Etiology.

Hitherto we have only treated of chronic ostitis in so far as it is dependent on periostitis. This is almost always the case in the hollow bones, for in them the cortical layer is not much disposed to become primarily diseased. The case is different with the spongy bones and bony parts; in them a chronic inflammation may arise independently, just as in the medullary cavity of a hollow bone there may occur a circumscribed chronic osteomyelitis, so that the cortical substance may become diseased from within. These cases are designated as *ostitis interna* or *caries centralis*. The symptoms of such a chronic inflammation, occurring deep in the bone, are in many cases very undecided. A dull, moderate pain, and a consequent slight impairment of function, often form the only symptoms. Swelling comes on later, and the disease may exist for months before we can form a certain diagnosis. But when we find severe pain on pressure, and œdema of the skin, and the periosteum participates secondarily in the chronic inflammation, we shall gradually be led to the correct diagnosis, the more readily if the disease be circumscribed, and perforation finally takes place, so that we may pass a probe through the opening deep into the bone, and find exactly what is the state of affairs. In many cases periostitis is for a long time the chief symptom of ostitis; the former may be so prominent that it appears to be the only disease, till, from the long duration, and from losses of substance from within, or lastly, perhaps, even by detachment of small pieces of bone, attention is called to the fact that the continued suppuration is due to disease deep in the bone. It has already been stated that chronic inflammation in bone first shows itself by the chalky salts becoming soluble. So far we have only studied cases where the disease was circumscribed and progressed inward from the surface. Now, imagine an ostitis developing in a spongy bone, as one of the tarsal bones, or in the diaphysis of one of the long bones, as in the lower part of the tibia, and the chalky salts disappearing from the bony tissue while the vessels of the medulla increase, and the medulla, infiltrated with wandering cells, gradually takes the place of the disappearing bony tissue. Here we have the picture of a pure *ostitis malacissans*, an osteomalacia inflammatoria or rarefying ostitis (*Volkmann*). In this affection the bones become very light, and the cortical substance very thin.

*Rindfleisch* has shown how the atrophy occurs in such cases; for he discovered that the chalky salts were first dissolved and disappeared as in lacunar corrosion. But while in the latter case the osseous tissue disappeared with the chalky salts, in the present case the tissue continues to exist for a time; the cases where every trace of bone inside the periosteum has disappeared in this way, show that the osseous tissue which has lost its salts is finally absorbed itself. But whether this is always the case, or whether it may again be impregnated with chalky salts and again become normal bone, is not known. Whether this variety of atrophy, which may correctly be termed halisteresis ossium (from ἅλς, salt, and στέρησις, robbing, *Kilian*), always runs the course shown in Fig. 76, is not fully investigated; possibly the chalky salts and the tissue might be absorbed at the same time. The fact that there is no sign of proliferation in the bone corpuscles of the tissue deprived of its chalky salts seems to prove that they are not disposed to proliferate.

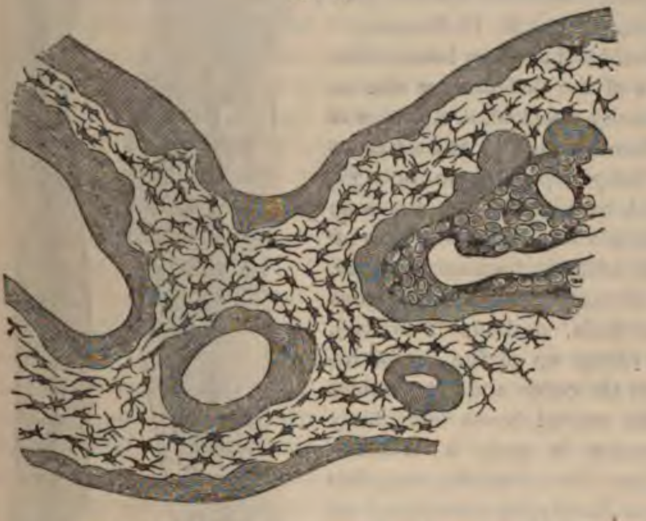
FIG. 75 A.



Ostitis malacissans: *a*, vertical section of the calcaneus, diseased anteriorly and posteriorly, normal in the middle; *b*, vertical section of the upper end of the tibia, quite porous.

So we have here a form of inflammation of bone in which its atrophy is a particularly prominent feature, and there is a very scanty formation of osteophytes, or this may be altogether absent. In the bone there is no regenerative process; the medulla, which is reddened from the great vascularity, usually contains fat, but is richer in young cells than the medulla of the bones of adults usually is, and hence more resembles the condition in childhood. The ostitis malacissans may remain in this state; should it slowly progress, it would lead to complete solution of the bone, till only medulla and periosteum remain, and the bone is so soft as to yield to any traction or pressure; but this is rare. According to my experience, it is just as rare for the medulla in these bones to suppurate or become caseous without some external cause; but this is sometimes induced by violent probing, dirty probes, bruising, or operations. Mild cases of this form of ostitis may recover by formation of new bone in the cavities of the old bone; while severe cases in marasmic patients are incurable and require amputation.

FIG. 76.



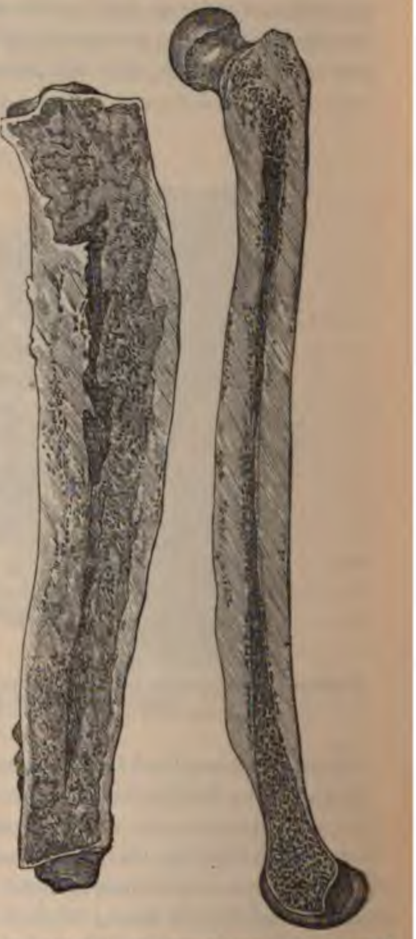
Disappearance of the chalky salts from the peripheral portions of the osseous framework in ostitis malacissans. Magnified 350. After *Rindfleisch*.

Ostitis osteoplastica is just the opposite of the above; we do not know whether the disturbance of nutrition by which it is started also begins with loss of chalky salts from the bone; the main effect of the disturbance is abnormal formation of new bone in the medulla and in the Haversian canals. When the disease occurs in the long bones, it generally attacks the whole bone at once, and even affects several bones at the same time. The result of this disease may be the complete filling of the medullary cavity, with a tolerably compact bony mass, the almost complete filling of the Haversian canals with bony substance, and generally also the formation of bone on the surface. Thus the entire bone becomes very heavy and denser than normal. This process is also termed *diffuse hypertrophy of the bone*, but more frequently *sclerosis ossium* (condensing ostitis, *R. Volkmann*).

Besides the hollow bones, other bones of the skeleton are also occasionally attacked, e. g., bones of the face and pelvis; in such cases the bony deposits are spongy, puffed, nodular, so that the bone acquires a resemblance to skin affected with elephantiasis; indeed, the diseases are very analogous (Leontiasis ossium, *Virchow*). The filling up of the diploë between the outer and inner tables of the cranial bones with bony substance is such a common change with advancing age, that it can hardly be considered as pathological, although it really belongs under this head.

The causes of sclerosis of bone as a primary disease are entirely obscure; in some cases syphilis may act as a cause, but the osseous formations occurring in this disease rarely attain such firmness as in sclerosis proper. The malady will rarely be recognized with certainty during life, because to the touch these bones present nothing more than a certain increase of thickness and a slight inequality of surface.

FIG. 77.



Sclerosed tibia and femur; the former after *Follin*, the latter from a specimen out of the Vienna Pathological Anatomical Collection.

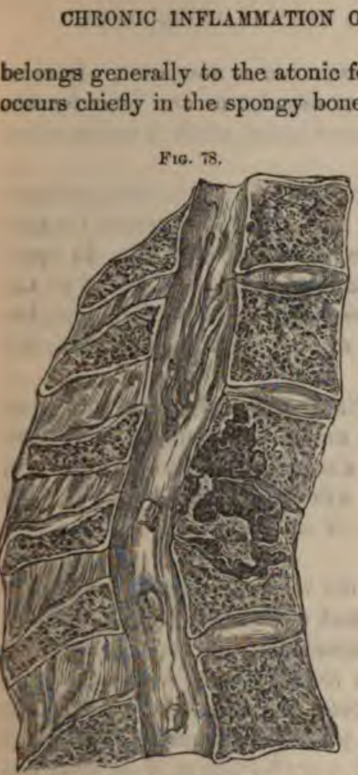
*Ostitis interna suppurativa circumscripta* usually begins in a hollow bone as osteomyelitis. The inflammation gradually extends to the inner surface of the cortical substance, which is dissolved, as we have already stated, and finally completely consumed at some point. In such cases pus may form quite early in the centre of the inflammatory new formation, and subsequently be evacuated. It is this disease that is especially termed *bone abscess.* The periosteum does not remain unaffected; it is thickened and new bony deposits form in this case also from the surface of the bone, which is not at first perforated but is irritated from within. The hollow bone is thus enlarged externally at the point where the abscess forms in it, and gives the impression of the bone being here pressed apart and inflated. It is difficult, indeed often impossible, to distinguish such a bone-abscess from a circumscribed osteoplastic periostitis, hence we should not be in too great haste to operate. This central caries may be accompanied by partial necrosis of certain portions of bone on the inner surface of the cortical substance, forming a *caries necrotica centralis.* Lastly, we have the worst cases, where chronic internal and external caries are accompanied by necrosis and by suppurative or osteoplastic periostitis. All these develop in one and the same hollow bone at the same time; abscesses appear at different points; with the probe we sometimes touch rotten bone, sometimes a sequestrum; in one place we enter the medullary cavity of the bone, in another only the surface appears diseased; the whole bone is thickened, as is the periosteum, and a little thin pus escapes from the fistulous openings. The macerated preparation of such a bone has a peculiar appearance; the surface is covered with very porous osteophytes; between these, here and there, we find necrosed portions which belong to the surface of the bone; some openings lead into the medullary cavity; if you saw through these bones longitudinally, you find the medullary cavity also partly filled with porous bony substance; the cortical layer has lost its even thickness, and it also is porous, so that it is only at some few points that it can be distinguished from the osteophyte deposits; in the original medullary cavity we find occasional round holes, and in some of these necrosed portions of bone. These bones are in such a state that their recovery cannot usually be expected, and either their extirpation or amputation of the limb is necessary.

In the *short, spongy bones* the case is somewhat different; in them, when there is proliferating, inflammatory neoplasia, solution of the bone with secondary suppuration comes on quite rapidly, although it is not an absolutely necessary result. There are cases of ostitis
the short spongy bones of the wrist and ankle, and especia
epiphyses of the hollow bones, where, without any decide
(which is usually caused by the resulting periostitis), the
tirely dissolved by interstitial granulations growing all
without any necessary accompaniment of the slightest

puration (*ostitis interna fungosa*). The result of such a solution of bone in these, or in other joints, is that by muscular traction the bones are displaced in the direction where the destruction is most advanced. And from this deformity we may judge approximately of the extent of the destruction. A short time since, I amputated a foot which was so distorted by such a destruction of bone, without any suppuration, on the inner side of the talus and calcaneus, that the inner border of the foot was greatly drawn up, just as in well-marked congenital club-foot, and the patient walked very insecurely on the outer border of the foot. A good-sized ulcer had also formed on the outer edge, which had latterly entirely prevented walking. I saw a similar case in the wrist-joint: A girl twenty years old had suffered for a long time from pain in the left wrist, without swelling of the soft parts; pressure on the wrist was very painful; gradually, without any swelling or suppuration, the hand became very much abducted; if the patient were anæsthetized, the hand could be returned to its normal position, and then it was found that part of the wrist had entirely disappeared. In the larger spongy bones, as the calcaneus, and in the epiphyses of the larger hollow bones, a central cavity, or a bone-abscess, may form, and this may be accompanied by a necrosis centralis. In the great majority of cases, however, the ostitis is accompanied by a purulent periostitis; this is particularly the case in the small bones of the wrist and ankle; these are so small that, when the periosteum becomes diseased, the disease readily extends to the entire bone and its articular surfaces, and that conversely primary disease of the bone quickly shows its effect on the periosteum and articular surfaces. In these cases also there is implication of the sheaths of the tendons and of the skin, which is perforated at various places by ulceration from within outward. In the hand the radius and ulna as well as the articular ends of the metacarpal bones may also be implicated, and in the foot the lower ends of the tibia and fibula, as well as the posterior ends of the metatarsal bones. The wrist and ankle joints are thus swollen out of shape; in many places thin pus escapes from the fistulous openings, and the bones of these joints are partly dissolved and partly replaced by spongy granulations, or else are entirely or partly necrosed. It is hardly necessary to tell you that the course of this form of primary suppurative ostitis also, in regard to vital relations, is just as variable as that of chronic periostitis, and that here also you see cases of a typical atonic, and others of a fungous variety, while there are a variety of cases between these extremes.

I must particularly mention one other form of chronic ostitis, viz., *ostitis with caseous degeneration* of the inflammatory neoplasia. You are already acquainted with this variety of chronic inflammation; it belongs generally to the atonic forms, with slight vascularization. It occurs chiefly in the spongy bones, and readily combines with partial necrosis; in the cheesy pulp which fills the cavity in the bone there are almost always portions of dead bone that have not been dissolved. The vertebræ, the epiphyses of the larger hollow bones, and the calcaneus, are the most frequent seat of this *ostitis interna caseosa.* This form is only recognizable in a few cases during life; we gradually arrive at the diagnosis of ostitis interna, but can only determine its special form in cases where the half-fluid caseous pulp is evacuated through an external opening. Lastly, we must not omit to mention that in rare cases, usually in the vicinity of caseous deposits, true *miliary tubercles,* small, at first gray, later cheesy nodules, come in the spongy substance of the epiphyses in the ankle-bones and vertebræ and induce solution of the bone and partial necrosis. A diagnosis of this true bone tuberculosis cannot be certainly made during life, we may only consider it as probable where there is marked tuberculosis of the lungs or larynx.

FIG. 78.



Point of caseous degeneration in the spinal column of a man.

For all forms of ostitis, which induce softening of the bone-substance, *R. Volkmann* employs the designation *rarefying ostitis.*

[We have now described a series of chronic processes, which may occasionally occur in the same bone, but whose natures are different. It is common to all for the normal bone substance to be dissolved and replaced by a soft, cellular neoplasia, except in the so-called sclerosis ossium, in which the neoplastic tissue is transformed into compact bone. Some of these processes are inflammatory, as gous ostitis, which leads to intraosteal development of granu and sometimes to abscess, caseous ostitis, etc.; but in so-call malacissans (rarefying ostitis), as well as in ostitis osteopla inflammatory symptoms are more obscure. While in the may sometimes be suppuration, in the latter it never the latter processes new development of vessels seems for reabsorption as well as for the formation of new bone. In rarefying ostitis the process is limited to solution of the bone by formation of medulla which is transformed to fat, while in ostitis osteoplastica it ossifies.

In the forms of ostitis which destroy the bone the final products which take the place of the bone are granulation-tissue (fungus without suppuration), bone-pus, caseous neoplasia, or fat. In opposition to these destructive forms stands osteoplastic ostitis as the sole productive form; at first it too partly dissolves the bone, but soon it forms new bone, not only to replace what has been lost, but even beyond the normal amount.

Bony tumors, growths, or deposits in the periosteum will occasionally disappear spontaneously or under some simple treatment. It is thought by some that they are due either to gouty, rheumatic, or syphilitic poison, and that they will generally be benefited by iodide of potassium or a mild course of mercury.

In children with hereditary syphilis we sometimes find multiple caries of the skull circular in shape and surrounded with a vascular network, within which, without suppuration, the bone is softened and deprived of its chalky salts; so that at these points the skull seems to be replaced by a translucent vascular membrane, while there is not a trace of osteoplastic periostitis, or any considerable infiltration of the soft parts. Such a skull looks as if several trephine holes had healed up, but not with bone.

Scrofulous diseases of bone may be induced by slight injuries, as was long since confirmed by clinical observation as well as by experiment. We have previously stated that acute infectious diseases have an influence in developing chronic ostitis; but it is doubtful whether contagion here, as in acute infectious osteomyelitis and tuberculosis, develops and spreads by micrococcus vegetation.]

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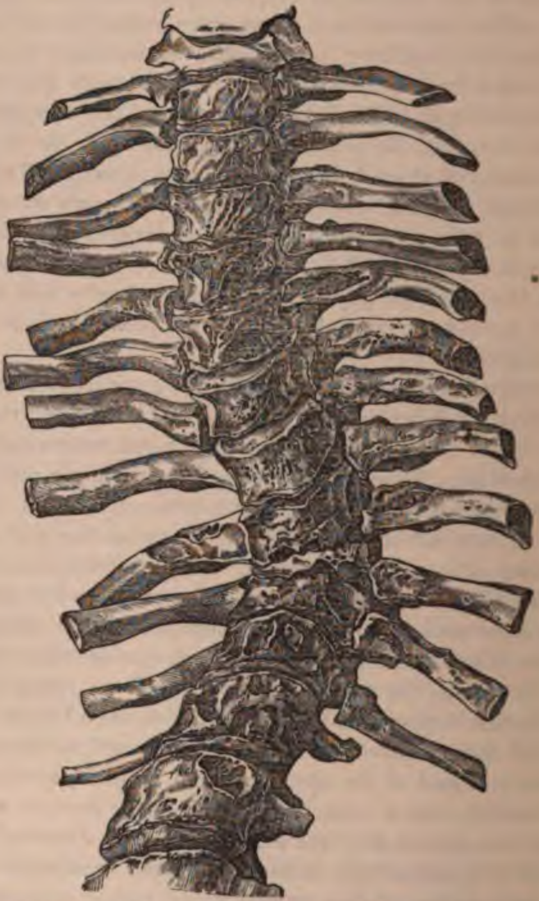
From the occasional remarks that I have made concerning the *diagnosis* of chronic periostitis and ostitis, you will have already seen that, after they have lasted a certain time, their recognition is not generally difficult, but that it is not always possible to state the variety and extent of any given case. There are two very important factors for the diagnosis in those cases that cannot be examined directly by the sound, viz., the *displacement of the bones*, which must result, in many parts of the body at least, from their partial solution, and *formation of abscesses*, which often accompanies it.

Carious destruction of the larger hollow bones rarely goes so deep as to cause a solution of continuity; where this might otherwise occur, it is often prevented by osteophytes growing on the outside while the destruction goes on within, so that the bone grows thicker at the point of disease. I have only seen one case where, from a perfectly atonic caries of the tibia of an old, decrepit person, the bone was at one point so far consumed that there were entire loss of continuity and spontaneous fracture; *post-mortem* examination showed that there was not a trace of osteophytes. The bone in Fig. 74 is also nearly eaten through. Complete destruction of the substance of the small hollow bones of the phalanges and metacarpi is not so rare; the scrofulous caries of these bones has from time immemorial been called *Pædarthrocace*, or spina ventosa, old names that only mean caries in the fingers or toes, with spindle-shaped enlargements. Should the bones be entirely destroyed by the fungous proliferation and partial necrosis of the small diaphyses, the fingers atrophy and are drawn back by the tendons so strongly that they represent misshaped rudiments of fingers.

Displacement of the spongy bones is far more frequent when they are destroyed. I have already spoken of this as occurring in the wrist and ankle bones, still, it occurs far more extensively in other bones; for instance, if the head of the femur and upper margin of the acetabulum are destroyed, the femur is gradually drawn up in proportion to the amount of destruction, and assumes the position that it has in upward dislocation of the hip. Similar dislocations occur in the shoulder, elbow, and knee, though there they are less remarkable. About the most noticeable are the dislocations in the spinal column after carious destruction of the vertebræ; if one or more vertebræ be destroyed by ostitis, the part of the spinal column lying above this point has no firm support, and must sink; but, since the arches of the vertebræ and spinous processes are rarely diseased at the same time, only the anterior part of the spinal column sinks in, and an anterior curvature results, and a consequent posterior projection, a so-called *Pott's boss*, thus named after the English surgeon, *Percival Pott*, who first accurately described this disease. In every anatomical collection you find preparations of this, unfortunately, rather common disease. The occurrence of such a boss is occasionally the sole, but tolerably certain, sign of caries of the vertebræ.

A second important symptom of destruction of bone, or caries, is the suppuration which accompanies many or most cases. The pus collects around the diseased bone; a cold abscess forms; the pus does not always remain at the point where it forms, but sometimes sinks deeper, particularly when it has displaced the parts from within outward, so that it reaches the loose connective tissue. The most frequent source of such sinking or *congestion* abscesses is the above disease of the vertebræ; as this most generally begins as chronic periostitis on the anterior side of the vertebræ, so this is the most

FIG. 79.



Destruction of the vertebral column by multiple periostitis and ostitis anterior. Preparation from the pathological anatomical collection at Basel.

common seat of the suppuration; the pus sinks behind the peritonæum, along the psoas muscle, and usually makes its appearance below Poupart's ligament, and to the inner side; it may possibly, but more rarely, take a different course, as backward. These congestion abscesses are of great diagnostic and of still greater prognostic value;

as a rule, they are bad signs; their treatment, of which hereafter, is one of the most difficult points in surgical therapeutics. In speaking of the sinking of pus, it is meant that, following the laws of gravity, the pus sinks mechanically; it will do so most readily where there is simply loose connective tissue present, and no opposition from fascia, muscles, or bone. But I must call your attention to the fact that this purely mechanical picture is only partly correct; for it is partly an ulcerative suppuration that progresses in a certain direction, which is only slightly influenced by the pressure of the pus; the abscess enlarges as it does in other cases; if the pus reaches a point under the skin of the thigh, perforation usually results, not from the mechanical pressure of the pus, but from ulceration from within outward, as in the opening of other abscesses; such a congestion abscess may last one and a half to two years before opening spontaneously.

We come now to the *etiology of ostitis and caries interna*, which we may treat very briefly, as the chief factors act here as in chronic periostitis, or in chronic inflammations generally.

It is, on the whole, rare for injury to induce ostitis chronica; but this may develop in the form of an osteomyelitis in one of the larger hollow bones, from severe concussion and bruising, with extravasation of blood in the medullary cavity; the same thing may occur from contusions of the bones of the wrist or ankle. But it is more common for such causes to induce acute disease, such as acute periostitis. If suppuration take place after contusion of the wrist or ankle, if the cartilage be destroyed and the suppuration extend to the bone, we may have fungous ostitis of the small spongy bones, and their complete destruction. Even healthy, strong persons may, from protracted traumatic inflammation of the joint, become so anæmic and cachectic that the disease will not go on to its normal termination, but becomes chronic.

Most frequently scrofula and syphilis are the causes of chronic inflammation of the bones; in scrofula, while the children are fat and well-nourished, the fungous forms predominate. In thin, badly-nourished, scrofulous children, on the contrary, ostitis with caseous degeneration and other atonic forms not unfrequently develop; both of the latter lead to partial necrosis. The most frequent seats of scrofulous ostitis and periostitis are the vertebræ, articular epiphyses, phalanges, and metacarpal bones; the jaw-bones and large hollow bones are rarely affected.

In syphilis, ostitis and periostitis osteoplastica are most frequent in the tibia and cranium; caries sicca fungosa also occurs, sometimes primarily in the diploë of the skull, sometimes after periostitis;

the sternum, palatine process, and nasal bones, are often affected; necrosis often follows syphilitic caries. Some recent authors, such as *R. Volkmann*, represent syphilis of the bone as something peculiar, under the name of *ostitis gummosa;* I acknowledge that certain combinations are particularly frequent, giving rise to typical pictures of the disease; but, anatomically, syphilis in the bone is nothing more than ostitis and periostitis. In many cases, even on most careful examination, we are unable to find any local or general cause for the existing caries, and I consider it better to admit this than to try with all our might to discover some dyscrasia.

## LECTURE XXXIV.

Process of Cure in Caries and Congestion Abscesses.—Prognosis.—General Health in Chronic Inflammations of the Bone.—Secondary Lymphatic Enlargements.—Treatment of Caries and Congestion Abscesses.—Resections in the Continuity.

Before passing to the treatment of chronic periostitis and ostitis, we must add a few remarks about the *process of cure* in caries, and about the *prognosis.* The first will vary somewhat with the activity of the process, as it does in ulcers of the skin. Supposing the process of proliferation in the new formation to cease, the interstitial granulation-tissue will gradually shrink together, and be transformed into cicatricial tissue. Considered histologically, this process consists of the retrogression of the gelatinous intercellular substance into firm, filamentary connective tissue, while the richly-developed capillary vessels are mostly obliterated, and the cells acquire the character of connective-tissue cells. If the caries was accompanied by suppuration, the latter gradually ceases, and the fistulæ close. If part of the bone had already been destroyed by the ostitis, and there was displacement, it does not disappear, but the loss of bone is supplied by a retracted connective-tissue cicatrix, and the dislocated bones are united in their false position by such a cicatrix; this connective tissue generally ossifies subsequently. The cicatricial union of two dislocated bones, as of two vertebræ, which have come into contact by the destruction of a vertebra previously lying between them, also ossifies, and thus unites the vertebræ firmly; the actual substitution of bone for any neoplasia to such an extent as to straighten the spine again, or entirely or partly to replace any other bone, never occurs in caries.

Should an atonic ulcer of the bone heal, it may do so in one of two ways: either any portion of bone that has become necrosed must be detached and thrown off, then by a rich development of vessels, a vigorous new formation must form from the walls of the defect, and when there has been a large excavation or abscess in the bone the entire cavity must be filled with granulations before recovery is possible—for a perfect cure these granulations must cicatrize and ossify, and to a certain extent the torpid ulcer in the bone must become proliferating—or else granulations arising from the healthy bone behind the diseased, necrosed portion dissolve the latter; at the same time the torpid process becomes proliferating, and thus leads to cicatrization. The defects in bones, for example, in the centre of a hollow bone, cannot decrease by contraction, which so much curtails healing in the soft parts, but must be entirely filled up by new tissue. This is the point that so often prevents recovery in caries, for the constitutional conditions at the root of the torpid form of caries are often so serious that it is not only difficult to arrest the advance of the ulceration, but is just as difficult to induce active new formation in the seat of disease. If we actually succeed in arresting the process of ulceration, fistulæ not unfrequently remain and continue for years, or never heal. Nevertheless, when the disease remains stationary, the fistulæ in the bone rarely do much harm. If you have a chance to examine such fistulæ anatomically in macerated bones, you will find that the holes leading into the bone are lined by an unusually thick, sclerosed layer of bone, just like old fistulæ of the soft parts, whose walls consist of a hard cicatricial substance. We have still to speak of the process of cure of chronic cold abscesses in certain diseases; usually, if not opened, these do not heal till the bone-disease is on the way to recovery. Then, if the cavity of the abscess be lined with vigorous granulations, as is rarely the case, the walls may unite immediately; but more frequently, when such an abscess ceases to increase, it is first contracted by shrinkage of its inner walls, and is thus gradually closed. For this to occur it is requisite that the process of destruction should have ceased on the inner wall, and that the tissue should be sufficiently vascular. If a cold abscess do not open, but remain subcutaneous, while the bone-disease recovers, most frequently a large part of the pus, whose cells disintegrate into fine molecules, is absorbed, while the inner walls of the abscess change to a cicatricial tissue, which, in the shape of a fibrous sac, contains the puriform fluids. Such pus-sacs often remain in this stage for years; unfortunately, complete reabsorption, or absorption to such an extent as to leave only a cheesy pulp, is much rarer than might be desired, and than is usually supposed.

In the *prognosis* of a case of caries, we have first to consider separately the fate awaiting the diseased bone, and the state of the general health induced by long suppuration of the bone and soft parts. Regarding the fate of the part diseased we have already said enough, having on the one hand described the process of destruction and its results on the parts around, and on the other the mode of the possible cure. Here I shall only add the remark that, in caries of the vertebræ, as we may readily see, the spinal medulla may be endangered, by participation in the suppuration, or by being so bent, by the inclination of the vertebræ, that its function is destroyed; thus we may have paralysis of the lower extremities, of the bladder, or of the rectum, from caries of the spine. Practically, this is rarer than might have been expected *a priori*, because the spinal medulla is considerably protected by the hard dura mater, and bears quite an amount of gradual curvature without impairment of its function. The state of the general health, the grade and variety of the febrile reaction, are of general prognostic significance. Chronic diseases of the bone rarely begin with fever; indeed, in many cases, especially when there is no local treatment, and the consecutive abscess is allowed to open spontaneously, the patient escapes fever altogether. But this perfectly afebrile course does not continue; if the patient has remained free from fever previous to the opening of the abscess, after this there is usually hectic fever, which is generally a remittent fever with steep curves, i. e., low morning and high evening temperature. The earlier large cold abscesses are opened the sooner the afebrile passes into a febrile state, and not unfrequently there is an intense, exhausting, continued remittent fever; the chronic ulceration then often becomes an acute inflammation, with great tendency to disintegration of the diseased tissue; after the thin, flocculent, but not badly-smelling pus is evacuated, there is occasionally sanious suppuration, which may be only temporary. In such cases pyæmia may be the winding-up of the whole disease.

It is difficult to state the cause of this change of course after opening of a cold abscess, why the chronic inflammation should so quickly change to an acute form. The common supposition is, that the entrance of air excites severe inflammation in the walls of the large abscess cavity, which were already disposed to disintegrate, and that the oxygen of the air is the especial cause of the decomposition. This view may be correct in many cases, but it is not the air itself or the oxygen that is injurious, nor is it always the organic germs contained in the entering air. But it is certain that sometimes puncture or any method of opening gives enough irritation to excite an acute, spreading inflammation of the badly-organized walls of the abscess. In many cases also infectious matters may be inoculated by the instruments or dressings. [In the *Medical News and Library*, July, 1878, Dr. *S. W. Gross* expresses his belief that the circulation and nutrition of the walls are disturbed by the withdrawal of the contents; more blood is sent to the sac, its surface becomes studded with granulations from dilated capillaries, and pyogenesis is increased. After evacuation he recommends compression by adhesive plaster and bandages, and keeping the parts at rest.] The possibility of the chronic process becoming acute in this way justifies the prognosis that opening of the abscess increases the danger. We may here add that the general health is first decidedly affected by the suppuration; caries fungosa, whether running its course without suppuration or with only a slight amount, is consequently less dangerous to life than caries atonica, with great tendency to suppuration and decomposition. This prognostic point is also based on good grounds, for, as we have previously stated, proliferating inflammatory new formations more frequently occur under comparatively favorable constitutional conditions. If the fungous proliferations break down quickly, if the suppuration becomes more profuse and thinner, it is a bad sign, a sign that the general health has also become impaired.

The strength is used up partly by the production of pus, partly by the fever, and is only partly replaced because reabsorption does not go on properly from the stomach, digestion is not good; this reacts again on the local disease, and thus the general and local state are most intimately connected. The smaller the carious spot, the less dangerous it is for the general health; still there are certain localities where it is more dangerous than elsewhere; thus suppuration of the vertebræ, with large congestion abscesses, is very dangerous, while caries of the phalanges, even if several be attacked, has little effect on the general health; there is great difference in the danger to life according to the joint and diaphyses attacked; caries of the hip, knee, or ankle, is far more dangerous than in the shoulder, elbow, or wrist. Of this we shall speak more particularly when treating of diseases of the joints.

The age is also of great prognostic importance in caries—the younger the patient the better hope of recovery; the older he is, the less hope: in caries coming after the fiftieth year, whether a sequent of periostitis or primarily as ostitis, the prognosis as to recovery is very doubtful, insignificant as the local disease may be at first; I do not remember ever to have seen caries in old persons so frequently as at Zürich. Lastly, the prognosis depends greatly on the constitutional disease to which the caries is due. Relatively, syphilitic caries is the most favorable, because we can treat syphilis the most successfully. In well-nourished children scrofulous caries also is rarely dangerous to life, as the scrofula disappears spontaneously, or after the use of proper remedies. But caries in atrophic scrofulous children is dangerous, because such children easily die of exhaustion. The prognosis in caries is most unfavorable where there is already pronounced tuberculosis; it very rarely recovers; the pulmonary disease generally advances rapidly and acute miliary tuberculosis develops in the serous membranes, and sooner or later terminates life.

The patient, dying slowly from chronic suppuration, gradually grows more and more emaciated, pale, and very anæmic, at last œdema of the lower extremities comes on; he eats less, and finally, after years of suffering, he dies of marasmus, often very slowly; sometimes he sinks to rest quietly; sometimes struggles for days with death. Formerly it was generally supposed that death in these cases was solely due to gradual exhaustion; but more careful examinations have shown that the exhaustion and impoverishment of the blood often have very palpable causes. For in these cases we often find the liver, spleen, and kidneys, in a state of fatty or amyloid degeneration (*Hyalinose, O. Weber*), a variety of degeneration which consists in the deposit in the substance of the organ, from the smaller arteries, of a peculiar material characterized by its lardaceous consistence, and by its reaction; on addition of iodine and sulphuric acid, it colors partly deep-reddish brown, partly dirty-brown violet, with a play of colors into green and pale red. Concerning the nature of this material there are various views, which you will find more detailed in the pathological anatomies. I shall only tell you here that the above reaction with iodine and sulphuric acid is similar to that of cholesterine, and that consequently *Heinrich Meckel von Hemsbach* believed that the fatty substance owed its reaction to the large amount of cholesterine it contained. Others thought that this material was allied to amylum, and hence *Virchow*, who held this view, called it *amyloid*. *Kuhne* subsequently showed that both of these views were untenable. The so-called amyloid is a peculiar substance, closely allied to albumen; it differs from albumen particularly by its insolubility in acids containing pepsin. From the mode of its occurrence this material is very interesting and noteworthy; it and fibrine are the only organic bodies we know that pass in fluid form through the vessels, and outside of these coagulate firmly in the living body, without the vital power of cells appearing necessary.

The saturation of the liver, spleen, and kidneys, as well as of the walls of the intestinal arteries and of the lymphatic glands, with fat, must naturally have great influence on the formation of the blood, and finally prevent it entirely; thus, in most of these cases death is caused by disorganization of the blood. Extensive chronic suppurations greatly predispose to fatty degenerations; hence, in patients with extensive caries we should carefully attend to this point, though frequently we cannot avert it. Besides tuberculosis and amyloid degeneration, which unfortunately not unfrequently combine, these poor patients are occasionally also endangered by the common forms of acute and chronic diffuse nephritis, or morbus Brightii.

I will also mention that, in chronic inflammation of the periosteum and bone, the proximal lymphatic glands often participate in the disease. As in acute inflammations the lymphatic glands are often infiltrated and excited to acute inflammation by material coming to them from the point of disease, so in chronic inflammations the same thing occurs and from the same cause. The lymphatic glands swell slowly, painlessly, but often enormously in the course of months and years; the tissue of their frame-work thickens, some lymphatic vessels are obliterated, while others increase in size; rarely it goes beyond this hyperplastic swelling; occasionally there are small abscesses and points of caseous degeneration.

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Now, after having examined chronic periostitis and ostitis from all sides, it is time to think of the *treatment*. In so doing, after having spoken of these diseases in their most varied extent and combination, we must again begin with simple chronic periostitis. The treatment should be at once general and local; in all cases where dyscrasial causes are evident, they should be chiefly treated, and on this point I refer you to what was said in the general consideration of these dyscrasiæ in the chapter on chronic inflammation. Therefore in this place we shall chiefly consider *local remedies*. Rest of the diseased part is the first and most general rule in the treatment of chronic inflammation of the bone; for movement, accidental blows, falls, etc., may change what would have been a mild, not injurious course, to an acute and dangerous one; hence, in most cases of disease of the bones of the lower extremities lying quiet is of the first necessity, in the upper extremities carrying the arm in a sling. This rest is particularly important in diseases of the bone near the joints; under such circumstances rest is often spontaneously resorted to because motion is painful. Some forms of fistulous caries become so quiet and painless, when suppuration externally begins, that motion has no effect on the diseased bone, and in such cases moderate motion may be allowed.

Elevation of the diseased part is a good adjuvant to the treatment, for it avoids venous congestion. This mechanical aid to the escape of the blood must not be undervalued.

When the first symptoms of chronic periostitis and ostitis begin.

treatment should aim at inducing resolution. For this purpose, powerful antiphlogistic remedies are of little use. The application of leeches or cups, the internal administration of purgatives, the application of bladders of ice, seem to me only beneficial in acute exacerbations of chronic inflammation; their action is always very temporary, and the employment of local bloodletting and purgatives may even prove injurious if often repeated. The repeated application of leeches and cups proves locally irritant, and may finally make the patient anæmic, and a continuance of laxatives exhausts his strength; hence we should employ these remedies sparingly, reserving them for the acute exacerbations. Recently *Esmarch* has very urgently recommended the continued application of bladders of ice in chronic inflammation. In cases accompanied by great pain, I have seen very good effect from this treatment; in other cases I see no true indication for their use.

[As the continued use of ice is quite troublesome, we may employ in its place the apparatus recently devised by *Leiter*, of Vienna, by which a constant stream of cold water is passed through flexible, thin, metal pipes, which fit close to the body. In most patients moist warmth quickly relieves pain and local swelling. Another important point is elevation of the affected part, to favor the return of venous blood, which is also aided by application of roller-bandages. In some cases cure is hastened by applying elastic bandages till blood is driven from the parts, leaving them on ten to twenty minutes, and repeating this daily. At first the pain is usually severe, but ceases and does not return after removal of the bandage.]

In these earlier stages massage may be carefully tried. You will accomplish but little by these therapeutic efforts; you will find that as long as adult patients can tramp around on their diseased bones, they will do so; when you tell the patient that it is not certain the disease will get well if he lies quiet a few weeks, but that it will last months or years under careful treatment, he will attend to his business as long as possible. If the existence of your patient's family depends on his daily work, his case is very hard. It is just as hard to keep children constantly quiet; a grown person must watch them all day. This is impossible, not only among the poor, but in large families among those in moderate circumstances. It is very easy to say the child must lie still several months, except when it is carefully taken into the fresh air in a wagon or laid in a shady part of the garden during fine weather; but if this has to be done for years, it is very expensive, for it requires the whole time of a careful adult nurse. This daily, hourly care for securing the best hygienic and dietetic conditions to a child with chronic disease, requires unusual patience and intelligence. Sacrifices are much more readily made for expensive medicines, or going to watering-places, to get the trouble over quickly. In such cases we must consider the circumstances, so as to secure the best thing possible ; we may order mechanical supports to keep the weight of the body off the bones. I give you these hints, so that you may not be too much disappointed in your future practice. You will often see that many chronic diseases which are not incurable never are cured, on account of some social reason.

Most frequently, at the very commencement of chronic inflammation of the bone, the resorbent and milder derivative remedies are proper : officinal tincture of iodine, ointment of iodide of potash, mercurial ointment weakened by the addition of lard, mercurial plaster, ointments made with concentrated solution of nitrate of silver, hydropathic dressings, and mild compression-bandages. With these remedies, and proper constitutional treatment, we make our first attack on the diseases in question, if they are just commencing, and occasionally we succeed in arresting them at an early stage. In the early stages of serous and moderately-plastic infiltration and slight vascular ectasia, the retrogressive changes either occur without leaving a trace of morbid change, or perhaps leave a moderate formation of osteophytes. In this stage, the treatment of syphilitic diseases of the bone by active antisyphilitic remedies is the most successful.

If the process progresses, and the caries runs its course without suppuration, we may continue with the above remedies, and in suitable cases, in otherwise vigorous persons, may combine with the above, derivatives to the skin, such as fontanelles, the hot-iron, etc. If the signs of suppuration begin, and abscesses form, you may continue the absorbent remedies for a time, in the hope of even yet inducing reabsorption ; it is true, this will not succeed in most cases, but the question will soon arise : Shall we open the abscess, or wait for it to open? On this point I give you the following general rule : *If the abscess comes from a bone on which an operation is impossible or undesirable* (as the vertebræ, sacrum, pelvis, ribs, knee-joint, etc.), *do not meddle with it*, but be thankful for every day that it remains closed, and wait quietly till it opens, for thus there will be relatively the least danger. When I have departed from this principle, I have always regretted it. I saw, with great pleasure, that *Piriogoff* said almost exactly the same thing. Experience has sufficiently shown that none of our operations, aiming at imitating the slow spontaneous opening of these abscesses, prove as little irritating as the slow perforation of the skin from within by ulceration. Various methods have been proposed for opening large cold abscesses, corresponding to the theories in regard to them. For time it was thought that the pus must escape slowly, in order prevent inflammation of the abscess-walls. To accomplish this, setons were introduced, and the pus allowed to trickle from the points of opening. Then it was claimed that, besides this slow escape of matter, the skin should be perforated slowly. For this purpose, a caustic was applied to the thinnest spot of the abscess, and a slough made, which gradually became detached, whereupon the pus slowly escaped. Subsequently it was supposed that we should carefully avoid the entrance of air, as this was the dangerous point; so a trocar was introduced, a portion of the pus was evacuated and the opening accurately closed, or the so-called subcutaneous puncture, according to *Abernethy*, was made, i. e., the skin over the abscess was lifted up, and a narrow-bladed knife was passed under it into the abscess, a large part of the pus was evacuated; then the knife was quickly withdrawn, and the skin allowed to go back into its original position, so that the puncture in the skin did not communicate directly with that in the abscess-sac, but the latter was covered by the skin; the cutaneous opening was carefully closed. Subsequently great importance was attached to placing the walls of the abscess in such a condition that the formation of pus should cease; it was thought that this could be done by injecting solutions of iodine after the pus was evacuated; this method was especially popular in France.

[*Leister* and *Volkmann* propose, after carefully disinfecting the field of operation, to open the abscess freely by a long incision, and, instead of merely emptying it, to scrape its walls and remove the useless granulations with a scoop, arrest the bleeding by ice-water [or hot water] or compression, wash the cavity with a two-per-cent. solution of carbolic acid, and dry with clean sponges. After this, disinfect with a six-per-cent. solution of chloride of zinc, which causes a slight slough that prevents absorption of the products of decomposition. Now apply short drainage-tubes at various points, sew up the incisions, and apply a *Lister's* dressing and a good compress over the abscess-cavity. By this treatment, first the relaxed, inactive granulations forming the walls of the abscess, which are undergoing fatty or cheesy degeneration, are removed down to the healthy tissue, every opportunity for stagnation or decomposition of pus is diminished to the lowest point, and the collection of more pus after the sudden reduction of pressure is impeded by compression, which favors the immediate adhesion of the walls of the abscess, that now resemble freshly-wounded surfaces. This treatment, which requires great accuracy and thoroughness, has excellent results.

A recent improvement on the above is the use of iodoform. For some time past, after scraping the abscess-cavity I have filled it with iodoform, then unite the edges, except where a couple of drainage-tubes pass out, and apply a regular *Lister's* dressing. The iodoform prevents decomposition inside the sac, and inflammatory complications, while strong healthy granulations spring up and hasten the cure. The general state of patients thus treated improves wonderfully, which is partly because in the abscess-cavity there is no true pus, but only a moderate quantity of sero-mucous secretion. It is still a question whether by the iodoform treatment we may perhaps treat successfully those cold abscesses which are still inaccessible to *Lister's* dressing. Unfortunately, there are other cases which, do what you may, will turn out badly, and, whether you have treated them expectantly or actively, the public will ascribe to you the lack of success; for the laity have no idea of the danger of a cold abscess, and judge of it as they would of an acute one.]

Recently a French surgeon (*Chassaignac*) has returned with great enthusiasm to the old setons; but, instead of these, he chose fine tubes of caoutchouc with perforated walls, so that the escape of the pus was greatly facilitated (Drainage, page 109).

[While the introduction of drainage is commonly ascribed by the French to their distinguished countryman *Chassaignac*, it is really of older date. It is said that *Benjamin Bell* in the eighteenth century used tubes made of lead for this purpose.

Tubes made by winding wire on a metal rod of suitable diameter placed in a lathe, so as to form a spiral, make good drainage-tubes; they are flexible, and permit the entrance of pus at any part, while injections pass through to all points of the wound.]

*Lister*, an English surgeon, particularly urges that in opening these abscesses the instruments and dressings should be previously disinfected with carbolic acid, and also that the entrance of air should be carefully avoided; his proceeding, like all previous ones, has enthusiastic advocates. It is not easy to decide on the value of all these methods; but, when such a number of remedies and methods are recommended, you may almost always decide that the disease in question is very difficult to cure, and that none of the remedies are suited for all cases. Let us briefly criticise the above plans of treatment.

A single evacuation of the pus usually has at first a tolerable result, if done slowly and carefully, whether with the trocar or subcutaneously with the knife, with or without *Lister's* carbolic-acid treatment. If the opening is nicely closed and heals up, there is usually no fever, but the abscess fills again very quickly; an abscess that probably took ten months to form, may fill again in ten days. This is also punctured; the opening again closes; the patient grows feverish; the pus again collects rapidly. A third, and perhaps a fourth or fifth, puncture is made, always in a new spot; the patient grows more feverish, the abscess is hotter and more painful; the patient looks languid and suffering. Now the points of puncture cease to heal, the previous ones open again, there is a continual escape of matter, and occasionally, in spite of all our care, air enters, especially when the walls of the abscess are rigid and do not collapse. Now there is a fistula, the fever is continued, and the subsequent course is most unfavorable, as we described it above. So far as my experience goes, the course is not much changed if the puncture be followed by injection of iodine. There is not much difference if you make the opening with a seton, with drainage-tubes, or by cauterization. I have seen nothing from any of these methods that in the least approximated the claims of their proposers.

It is true this unfortunate course may be run if you do nothing to the abscess but leave it to itself and await its opening; but then all progresses more mildly and slowly, and fever comes on later. Recoveries take place under all these modes of treatment, but I think there are more recoveries, and certainly fewer deaths from pyæmia, under the expectant treatment. I am satisfied that where recovery has followed injections of iodine, drainage, etc., it would also have occurred had the course of the disease not been interrupted; we cannot accept the assertion that a case would have run its course thus and so, if this and that had not been done. Summing up my own experiences, I can assure you that, of very many cases of large congestive abscesses along the spinal column, artificially opened, I know very few that ran a favorable course; the others were only hastened to their end. Hence I again repeat the previous assertion, that these abscesses, especially congestive abscesses from caries of the vertebræ, are a *noli me tangere*. In such cases it is indeed frequently very difficult to wait; in private practice, especially, the patients become impatient; the surgeon is urged to do something, it is cast up to him that he does not try any thing; the public firmly believes that, if the pus was only out, recovery must follow. The surgeon also at length becomes weary; it is trying to look on from week to week as the abscess increases; all local and constitutional remedies are exhausted, and finally the surgeon departs from his principles and makes an opening; at first all goes well, but this does not continue; you already know the subsequent course.

The case is somewhat different when we have to deal with *small abscesses* originating in disease of *bones of the extremities;* in suppurations connected with the larger joints, we also willingly postpone opening; we shall speak of this hereafter, under diseases of the joints. In cold abscesses from the diaphyses delay is not of much avail; here I rather consider an early opening as proper, except in syphilitic gummata; in these cases there may be reabsorption, even after there is evident fluctuation, and in markedly tuberculous or debilitated persons, in them no operative interference is indicated, and opening the abscess would only induce profuse suppuration, without doing any good. In the other cases I am in favor of opening the abscess freely, to obtain a clear view of the variety and extent of the disease; under these circumstances the reaction is insignificant, frequently there is no fever, often there is moderate fever for a short time. Let us suppose a chronic periostitis with caries superficialis of the diaphysis of a hollow bone; an abscess has formed and been opened; the wound is at first dressed with charpie, and we then wait to see what appearance the surface of the ulcer will assume. The local treatment should be modified according as the ulcer is proliferating or accompanied by breaking down of tissue, and I should only be repeating, were I to refer again to the proper remedies. The treatment may be aided by local baths, which we may render slightly irritant by the addition of potash or tincture of iodine. Wet compresses, cataplasms, charpie-wads wet with various fluids, serve as dressings. The subsequent course will show more and more to what extent the bone-disease depends on the general health. If the patient be a weakly, tuberculous individual, all local remedies are in vain; if the general health be good, you may even resort to energetic local treatment. If the ulcer does not improve under milder remedies, you may apply the hot iron; should this be followed by formation of strong, healthy granulations, it is a favorable sign, even if there be necrosis of the carious portion of bone. In other cases we abandon all idea of inducing healing, and cut out the entire affected part. For this purpose there are various forms of cutting forceps and saws; I prefer detaching the diseased bone with scrapers, gouges, and hammer, to all other methods. If the ulcer of the bone has been cleanly cut out, and the general health be tolerably good, it is to be hoped that the wound of the bone made in the operation will heal normally by healthy granulation and suppuration, as other wounds of bone do. Should the caries affect a small bone, it may be proper simply to extirpate it, to arrest the process at once. If the case be one of ostitis interna, caries centralis of a hollow bone, or of a large, spongy bone, such as the calcaneus; if severe pain and other previously-mentioned symptoms of bone-abscess gradually appear, it may become proper to chisel out the bone, or open the cavity of the bone and let out the pus; but I only advise this operation when you are sure of your diagnosis, for it is no slight injury to a patient to have a healthy medullary cavity opened. Very acute osteomyelitis with its often dangerous results, may arise from untimely interference, while a similar operation on a diseased bone is not usually very serious. In other cases you will await the spontaneous opening of the abscess through the bone; then you may use a probe, and judge accurately of the state of affairs. The obstacles to the healing of such excavations in the bone have been previously mentioned; should the process remain stationary for a long time, it may be best to enlarge the opening in the bone, expose the abscess, and remove its walls; this will be the more necessary if there are any small necrosed portions of bone in the abscess-cavity which prevent its healing; that is, if the case be one of caries necrotica. But all these manipulations are only indicated if the general health be good; if there be advanced tuberculosis or marasmus, and the disease will necessarily prove fatal, no surgeon would wish to do an operation which can only prove successful when the local changes in the new wound of the bone go on normally. These operations, part of which, at least, may be classed among the *partial resections in the continuity*, have lost their cruel and terrible appearance since the introduction of chloroform, by whose aid the patients escape feeling the chisel, hammer, and saw.

In those cases where the caries is so extensive as to affect the whole thickness of a long bone, we might think of sawing out the entire diseased part. This case is very rare, and such operations are of extremely doubtful benefit. We might, it is true, saw out a piece from the middle of the fibula, radius, or ulna, from the metacarpal or metatarsal bones, without greatly impairing the function of the extremity; but, should we do the same for the humerus, femur, or tibia, and recovery take place, the function of the extremity would, at most, only be partially restored by aid of an apparatus; in the lower extremity an artificial leg would be of more use than a leg that had lost a considerable portion from the continuity of the bone. It has been thought that the periosteum, detached from the bone before it is sawed, and left in the wound, would form new bone; but after operations for caries this regeneration of bone is very scanty, so that we cannot count much on it. Moreover, caries is the rarest indication for these total resections in the continuity.

Lastly, in regard to those cases which are on the whole rare, where a hollow bone is diseased throughout with periostitis, external and internal caries, partial internal and external necrosis, there can only be a question of *extirpation of the entire bone*, or amputation of the affected limb. Cases of extirpation of the entire ulna or radius occasionally turn out well; extirpations of the first metacarpal bone are often successful. I also know of a case where the whole humerus was removed, leaving behind the thickened periosteum; but the patient died a few months after the operation from some internal disease, morbus Brightii, if I mistake not, so that no decision could be made about the usefulness of the extremity; in spite of the absence of the humerus, the hand might have been of service, which of itself would have been a great gain to the patient. Caries of the short, spongy bones, and of the articular epiphyses, is so intimately connected with diseases of the joints that we shall discuss it hereafter.

The state of general marasmus that finally occurs from diseases of the bone, with extensive suppuration, is to be treated on general principles. We should try to prevent its occurrence, or at least ward it off to the utmost. It is the physician's duty to preserve life as long as possible. It is also his duty, even in a patient almost certainly dying, to give him every thing that can keep up his strength. Nourishing, tonic, strengthening diet is to be given from the time the first symptoms of emaciation show the failure of nutrition; later it is of no use. In children and young persons the inexperienced physician may readily be deceived as to the strength, and you will hereafter see that patients in a very bad state, emaciated to a skeleton, and excessively anæmic, pick up wonderfully and unexpectedly on amputation of the diseased limb, which seemed to be consuming their life; of course benefit could rarely result from resection under such circumstances. How far it is safe to carry the principle of preserving the limb by sawing out the diseased portion of bone can only be judged of in individual cases, and then only approximately.

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## LECTURE XXXV.

Necrosis.—Etiology.—Anatomical Conditions in Total and Partial Necrosis.—Symptoms and Diagnosis.—Treatment.—Sequestrotomy.

GENTLEMEN: We have already frequently spoken of "necrosis," and you know that by this term we mean gangrene of the bone, death of a bone, or part of a bone. I have also told you that the dead portion of bone is called a *sequestrum*. You also know that necrosis may result either from an acute process, or accompany the process of ulceration as "caries necrotica."

As in death of any part, cessation of circulation is also the immediate cause of necrosis, while cessation of nervous activity does not induce it, although a disturbance of nutrition, an atrophy of the bone, is occasionally seen in paralyzed parts. Necrosis may be due to various causes; we shall briefly group them together:

1. *Traumatic influences.* Among these are severe concussions and injury of the bones, even without external wounds. The course is as follows: As a result of the above injuries there are extravasations in the medulla of the bone, also into the spongy bones, perhaps also in the compact bony substance, and occasionally under the periosteum. If these ruptures of the vessels be so extensive that their results cannot be removed by collateral circulation, which is of difficult establishment in bone, part of the bone will no longer contain any blood; this will die, and, according to circumstances, we may have central, superficial, or total necrosis (the latter occurs most readily in the small bones). The portion of dead bone remains in the organism as a foreign body, but still continues in continuity with the healthy bone; the solution of the sequestrum, by liquefaction of the bone-substance in the border of the living tissue, has been already explained (page 232). Another mode of injury is exposure of the surface of the bone, or sawing through a bone, by which the sawed surface becomes the surface of the bone; in complicated fractures a piece of bone may be so denuded of soft parts, and thus robbed of its circulation, that it becomes necrosed. We have also explained why the exposed bone or sawed surface does not always become necrosed, but that the bone may, like the soft parts, immediately produce granulations. Nevertheless, after the above injuries, superficial or partial necrosis is common enough, either because extensive clots form in the ends of the injured vessels of the bone, or because the vessels are compressed and suppurate on account of the acute suppuration in the Haversian canals.

2. *Acute periostitis*, *ostitis*, and *osteomyelitis*, are very frequent causes of occasionally extensive and especially of total necrosis of the hollow bones. In suppuration of the periosteum the supply of blood to the bone, by vessels passing through the periosteum, is cut off, and the suppuration is propagated through the Haversian canals to the medullary cavity; if the latter also suppurates, necrosis is inevitable, and will extend as far as the inflammation did. The same results will occur in primary acute ostitis and osteomyelitis with secondary periostitis.

3. *Chronic ostitis* and *periostitis* may combine with necrosis, for,

just as in the acute processes, suppuration, change of the inflammatory new formation to detritus or caseous matter, extends into the bone, and so impairs its circulation that part of the bone is no longer nourished and must necrose; atonic forms of caries induce necrosis more readily than the fungous forms, as has already been stated.

The necrosis that is supposed to occur after thrombosis or embolism of the chief trunk of the nutrient artery of a bone appears to be of more theoretical than practical importance. This variety of necrosis has hardly been proved by dissections on man; it is, moreover, very improbable, because the arterial supply, in full-grown bones, comes from so many sources that stopping one of the many afferent branches does not suffice to completely arrest the circulation in any considerable portion of bone. Although the collateral circulation in bone cannot, from mechanical causes, be greatly facilitated by dilatation of the vessels, and hence in capillary stasis there is always danger of partial necrosis, as already stated, still the connection, arrangement, and regular distribution of the capillaries, even in the firm cortical substance, are such that when the afflux is interrupted from one source it may easily come from another. In bone there are no defined capillary net-works and capillary groups as in the skin, but all the capillaries are intimately connected in all directions, as in the muscles.

The experiment of inserting a peg into the foramen nutritium in the upper part of the tibia of rabbits has been tried, and it has been followed by necrosis around the peg. I have made this experiment and obtained the same result by inserting the peg at any other part of the bone, and hence I believe that this experimentally-induced necrosis depends only on the variety of the injury to the bone.

It will be proper now to study more accurately the anatomical course of necrosis, especially of that coming after acute periostitis and osteomyelitis. I have already told you, on various occasions, when treating of the healing of fractures and of chronic ostitis and periostitis, that the vicinity of such collections of pus is almost always affected in such a way that osteophytes form on and in the bone; their development is greatly influenced by the periosteum, and also by the surrounding parts (where they form after fractures). While solid healing is due to this new formation of bone after fractures, in chronic ostitis and periostitis it is more an accidental product of irritation, which subsequently has no further significance. The same thing is true in superficial necrosis. When, from new deposition of osteophytes around the sequestrum, the bone becomes more dense around the point of disease, whether this be exfoliation of one of the cranial bones, or a sequestrum from a sawed surface, it has no further practical importance. It is different in complicated fractures: when the broken ends or nearly loose fragments of bo become necrosed, the formation of new bone in the vicinity may n only induce future firmness in the bone, but the sequestrum may entirely enclosed by the new bone, and it may be necessary to rem it by operation. But this formation of new bone is most import in total necrosis of entire diaphyses; it is intended to replace the bone which dies. This very important process, which is so wonderfully accomplished by Nature, we must now study more carefully. Let us suppose an acute total periostitis and osteomyelitis with necrosis of the diaphysis of the tibia. The entire periosteum and medulla have suppurated; within the bone the pus falls to detritus, or actually putrefies; the pus from the periosteum has perforated the skin at various points, the circulation in the diaphysis has ceased; the entire diaphysis is a sequestrum. A longitudinal section gives the following appearance (Fig. 80):

FIG. 80.

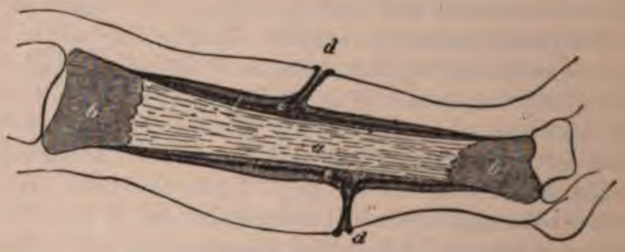


Diagram of total necrosis of the diaphysis of a hollow bone.

*a*, the sequestered bone; *b b*, its upper and lower extremities; *c c*, pus surrounding the sequestrum; *d d*, where it has perforated externally. The darkest layer, *e e*, is the wall of a large abscess-cavity, which consists of tissue (connective or tendinous tissue, or even of muscle), infiltrated with plastic matter, and on its inner surface, which lies next the sequestrum, like any abscess-cavity, it has a granulation-layer, which constantly produces new pus. I will mention at once that this view, as in acute periostitis, differs from that of other surgeons and anatomists, because they suppose the tendinous portion of the periosteum is lifted, like a vesicle, from the bone by the pus; this is incorrect, because the tendinous portion of the periosteum is not sufficiently elastic to be quickly elevated like an epidermis vesicle, and because this elevation would fail to occur at those points where there is no periosteum, i. e., where tendons are attached to the bone; but the latter is not the case. The inflammation and suppuration begin partly in the surface of the bone, partly in the softer parts of the periosteum, in its outer layers; the tendinous portion participates but little; indeed, it is mostly destroyed. In proof of this I have very decided anatomical evidences. The anatomists and surgeons who believe in the elevation of the periosteum consider the shaded layer, *e e*, as infiltrated, thickened periosteum; this is only conditionally true: it may happen that part of the periosteum does not suppurate and enters into the composition of this layer; however, other adjacent parts may also be so indurated by plastic infiltration as to form a firm abscess membrane, as is often seen in abscesses of the soft parts. Whoever maintains the exclusive power of the periosteum to produce bone will, on theoretical grounds, regard this layer, *e e* (where bone s subsequently formed), as thickened periosteum. But, in the formation of callus, after fractures, we have already seen that bone in considerable quantity may under certain circumstances be produced in other soft parts lying near the bone, and hence we are not obliged to demand periosteum in this thickened layer of the abscess.

But we are going on too rapidly. Let us return to our example. The pus-cavity around the sequestrum cannot close till the latter is out of it; but this remains attached at both ends. You already know how the detachment is effected: at *b b*, in the edges of the living bone, there is an interstitial proliferation of granulations, by which a slight amount of bone is consumed, so that at last the osseous substance is entirely replaced by soft granulations at these ends; this completes the detachment of the sequestrum (see page 232); the granulations forming here break down somewhat, soften to pus, and then the sequestrum lies loose in a pus-cavity, which is filled with proliferating granulations. In the thick hollow bones this detachment of the sequestrum requires a long time, usually several months, sometimes over a year; up to this time the pus has escaped from the places where it had perforated the skin; if, during this time, you introduce a probe through the openings, you may usually feel the smooth surface of the diaphysis. But, during this process of detachment of the sequestrum, something else is generally going on in the immediate vicinity, to which we shall now turn our attention. In the thickened layer of the pus-cavity, *e e*, new osseous tissue has formed regularly around the sequestrum longitudinally; this ossification has also continued to the part where the thickened layer again joins the periosteum of the epiphysis and the capsule of the joint, so that the bone-capsule is intimately connected with the epiphysis above and below. The longer the sequestrum remains in the cavity, the more the bony envelope increases in thickness; in time it becomes very thick; in the course of years, if the sequestrum does not come out, it may be over half an inch thick; at first, it consists of porous bone, but subsequently is more compact and stronger. A regular cast has been formed around the sequestrum, just like we should make of plaster of Paris if we wish to mould an object; this cast, however, has several openings, especially where the pus escapes; their closure is prevented by the constant flow of pus. The above picture (Fig. 80) has now changed to the following (Fig. 81):

FIG. 81.

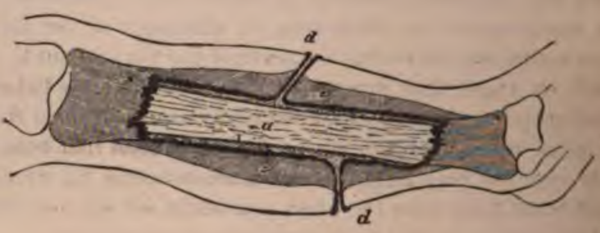


Diagram of total necrosis of the diaphysis of a hollow bone, with a detached sequestrum and new bony receptacle.

The sequestrum *a* is detached and bathed in pus, which is secreted from the granulations above mentioned; *d d*, the fistulæ leading into the pus-cavity (they have received the name *cloaca*); *e e* is the bony envelope derived from the ossification of the thickened abscess-wall, the so-called bony receptacle. This thickening now progresses regularly, if the irritation caused by the sequestrum continues. Let us now suppose that the sequestrum escapes from its case (as happens occasionally—of this later), then, although all the bone of the diaphysis is lost, there is no disturbance of function, for the newly-formed bony envelope supplies the place of the bone that has been lost.

FIG. 82.

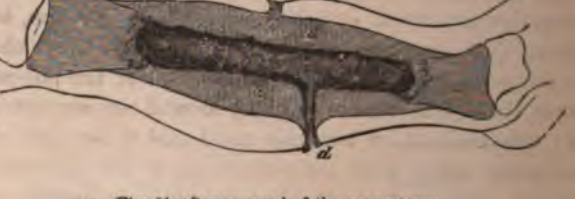
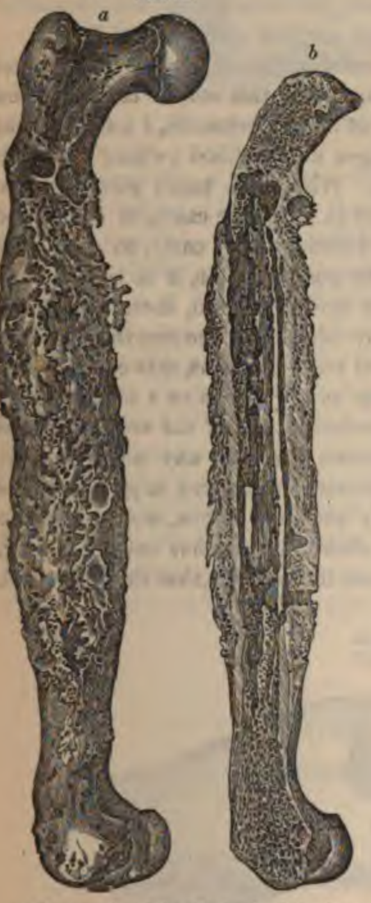


Fig. 81, after removal of the sequestrum.

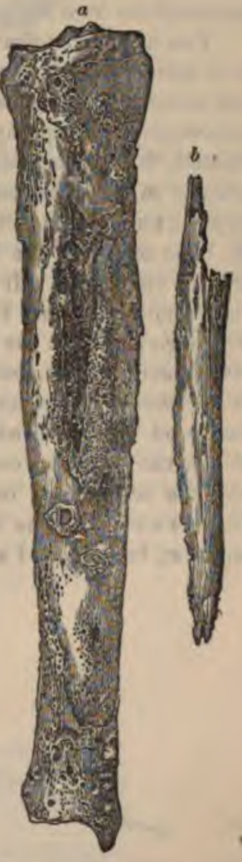
Now, what happens? Will the cavity in which the sequestrum may continue to suppurate? No; if every thing goes on normally, this cavity, like other cavities due to central caries, fills with granulations; these granulations ossify, and the bone is completely restored, at least as regards its form; observation has not yet determined whether the medullary cavity again forms in such cases as it does after the healing of fractures, but from analogy this is not improbable. After removal of the sequestrum, the healing of these cavities often requires months and years, sometimes it is never complete, especially

Fig. 83.



a total necrosis of the diaphysis of the femur, with extensive bony case replacing the dead portion of bone; several good-sized openings lead through this bony case to the sequestrum within.
b, longitudinal section of the same preparation.

Fig. 84.

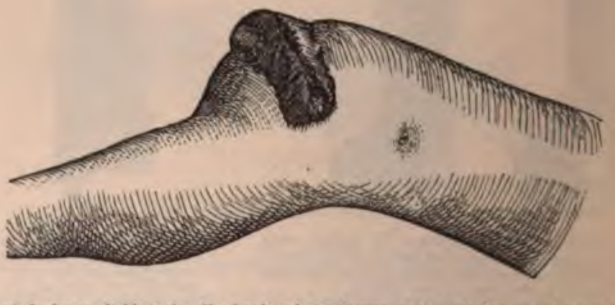


a, tibia of a young man after total necrosis of the diaphysis; about two years previously I had removed the sequestrum, *b*; the cavity has almost filled with osteophytes. The patient died from a carbuncle.

if the individual affected be constitutionally diseased, or becomes from the continued suppuration accompanying the process. In th long-continued suppurations from bone, albuminuria not unfreque develops, although of rather mild form. I do not know whether may in time spontaneously disappear after the cavity in the bone h healed; it would be interesting and of prognostic importance to co observations on this point. After removal of the sequestrum, the thickening of the osseous envelope ceases, and the process of ossification establishes itself in the cavity filled with granulations. What I have just demonstrated to you in diagrams, you here see in these beautiful preparations from the anatomical and surgical collection of Zürich.

You now know the ordinary normal course of a necrosis. I must next introduce you to some deviations from this normal course. You will remember that, when speaking of acute periostitis, I told you that occasionally the epiphyseal cartilages also ossified (where they still existed, that is, in young persons). When this takes place simultaneously in the upper and lower ends (a very rare case), of course the sequestrum will be detached, and detached very early, so early that no bone can have yet formed in the pus-cavity, or, if it has, it must still be very weak. If the bone be now extracted, there is nothing yet formed to replace it, nor does any thing form, because the irritation which gives rise to the production of bone is absent, this cause of irritation being the sequestrum, as long as it remains as a foreign body in the bone; hence, under these circumstances, if the sequestrum be extracted early, the extremity becomes boneless and unserviceable. When the epiphysis cartilage suppurates at one end, e. g., the lower end, the sequestrum remains firmly attached above, and the breaking down of the bone must go on slowly as in other cases; it may, however, happen, as I saw in one case in the thigh, that the lower end,

Fig. 85.

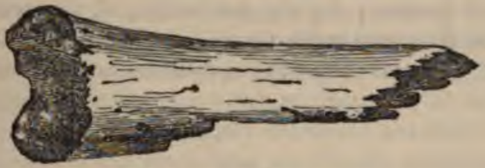


Necrosis of the lower half of the diaphysis of the femur, with detachment of the epiphyseal cartilage, and perforation of the skin.

loose in the epiphysis cartilage, presses strongly against the skin from within and gradually perforates it, so that it appears externally; the lower epiphysis of the femur was at the same time drawn up by the muscles, so that the appearance was as follows (see Fig. 85).

The sequestrum, subsequently removed, had the following form (Fig. 86):

FIG. 86.



The body extracted from Fig. 85.

The formation of bone was strong enough to carry the body; subsequently, under chloroform, the knee was straightened, and perfect recovery resulted. I saw a perfectly similar case affecting the lower end of the humerus. In both cases, as is usual in necrosis near the joints, the joint had suffered severely, and became quite stiff. Still, even without early detachment of the sequestrum from softening of the epiphyseal cartilages, under circumstances which we do not accurately know, the formation of bone may be very feeble, so that, after the detachment, the new bone is not firm at some point, but is quite flexible, whereby we have a pseudarthrosis of the new bone; I have seen two cases of this kind: one of these I cured completely by occasionally driving ivory plugs into the weak part of the newly-formed bone, thus constantly stimulating the bone to new production; the object was attained in the course of eight months, and the patient, then twelve years old, now walks like a healthy person.

FIG. 87.

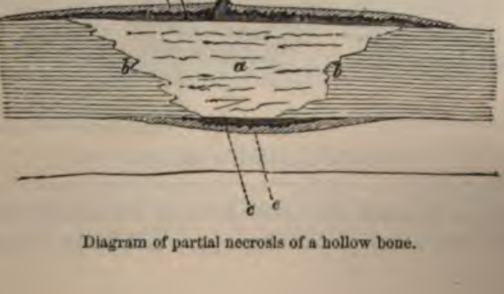


Diagram of partial necrosis of a hollow bone.

*Partial necrosis* of the diaphysis is more frequent than the above complete necrosis; this may either affect the entire thickness, or only half the circumference, according to the extent of the osteomyelitis and periostitis. You may readily apply what has been said to these partial necroses. Here is an example: suppose a periostitis of part of the diaphysis of one femur and subsequent necrosis; the circumstances may assume the following shape (see Figs. 87 and 88): *a*, sequestrum; *b b*, its borders; *c c*, the pus-cavity; *d*, the perforation outward; *e e*, the thickened ossifying wall of the pus-cavity.

A few months later (Fig. 89); *a*, detached sequestrum, which is to be removed; *e e*, newly-formed bone-tissue as substitute for the piece of bone that is being lost; of course, the newly-formed bone covers the sequestrum anteriorly, but, as in Figs. 80, 81, and 82, must be left out to expose to view the sequestrum.

FIG. 88.

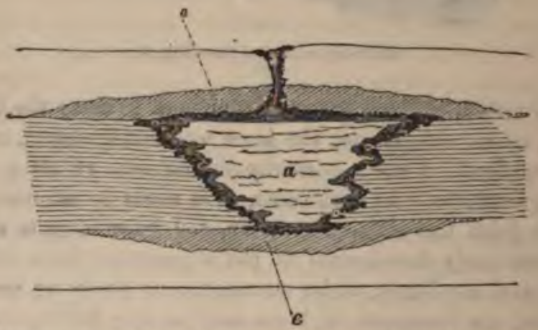


Diagram of Fig. 87 in the later stages, with formation of new bone.

FIG. 89.

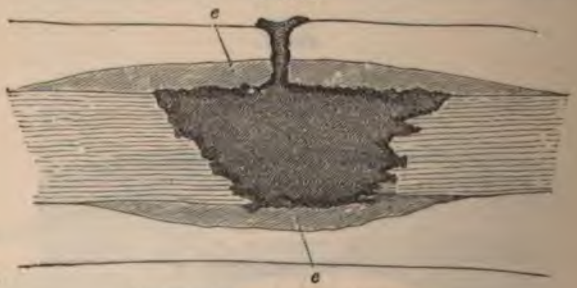
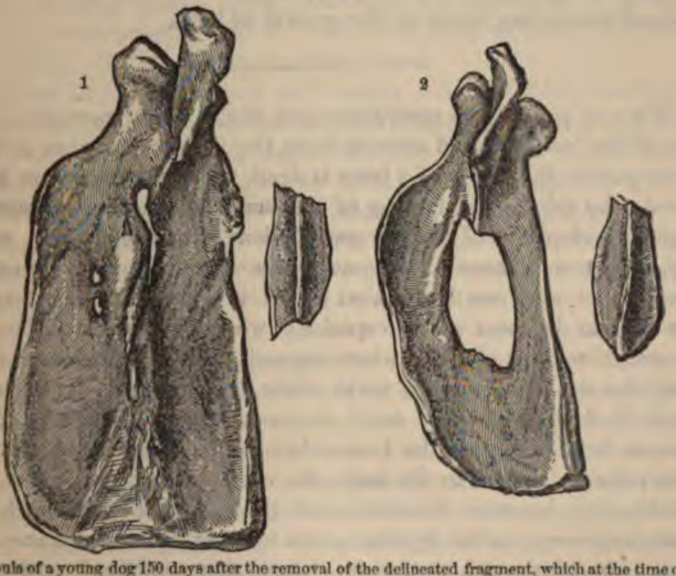


Fig. 88, after removal of the sequestrum.

The changes that we have now become acquainted with may also be applied to *necrosis in flat and spongy short bones;* but at the same time we must remark that in necrosis of these bones the new formation is much less, often entirely wanting, because the inflammation here is particularly of constitutional origin, and hence occasionally deviates from the normal course; as a rule, the inflammatory neoplasia in necrosis of the spongy bones soon assumes the ulcerative character, and then the formation of new bone is but slight; moreover, acute, non-traumatic periostitis is something very rare in spongy bones.

Extensive necrosis may even occur after originally pure ossifying periostitis and ostitis, in case the newly-formed ossific deposit is reabsorbed, suppurates and decomposes at the point of its attachment to the diseased bone; this gradually affects the nutrition of the bone; it often continues to live for a long time in the medullary cavity, or rather leads a half existence between living and dying; this variety of periostitis and necrosis occurs especially in the maxillary bones after chronic poisoning by phosphorous fumes, a disease peculiar to workers in match-factories. I cannot enter more minutely into this phosphorous periostitis and necrosis, which has many noteworthy peculiarities, because it would be necessary to load you with too

Fig. 90.



1. Scapula of a young dog 150 days after the removal of the delineated fragment, which at the time of the resection formed part of the fully-ossified portion of the scapula; the articular surface, edges of the cartilage, and the carefully-detached periosteum, were all preserved. The growth of the bone was unimpeded, and there was almost complete regeneration of the resected portion.
2. Scapula of a young dog of the same litter, 150 days after an operation performed the same day as the above, and in the same manner, except that the periosteum was removed. The growth was impaired, and the resected portion was not regenerated.

many details, which would now confuse you. If you bear in mind the above-described course of necrosis in the hollow bones, you will have the opportunity of learning in the clinic all the deviations that may occur in any case, from peculiar circumstances, for necrosis is a relatively frequent disease of the bones.

I cannot leave the anatomy of necrosis and the regeneration of bone accompanying it, without mentioning an excellent French worker who has spent many years in the study of the osteoplastic power of the periosteum, and has nobly carried forward the previous works of *Troja*, *Flourens*, *B. Heine*, *A. Wagner*, and others, on this subject: I mean *Ollier*, who, with untiring zeal, has pursued this study experimentally and clinically, and has closed it up for a long time; I have repeated part of his experiments, and can only confirm the idea that under certain circumstances, in young animals, preservation of the periosteum decidedly favors the reproduction of bone. In the course of these lectures I have already stated my opinion regarding the osteoplastic power of human periosteum, especially as compared with other soft parts surrounding the bones, and hitherto I have found these views confirmed by every new experience.

Quite recently *J. Wolff*, who was already distinguished by his careful provings of *Ollier's* experiments, has advanced some entirely new and interesting views on the growth of bone.

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We now pass to the *symptoms* and *diagnosis* of necrosis. Disease of the bone is called necrosis from the time it becomes evident that a part or the whole of a bone is dead, till the sequestrum is removed; the subsequent healing of the cavity in the bone is usually a simple development of healthy granulations with suppuration, which may, it is true, assume an ulcerative character. Now, the question arises, How shall we know that a part is necrosed? This may be very simple in some cases, especially where the necrosed bone is exposed, that is, in all cases where necrosis follows uncovering of the bone; the dead bone looks quite white, but in some places it becomes blackish, like other dried, necrosed parts. Gangrene of the bone, as far as regards the bone-substance, may remain as dry gangrene; the soft parts in the bone, the vessels, connective tissue, and medulla, may, however, like other soft parts, be attacked by dry or moist gangrene; perfect dryness occurs in most cases where the bone is uncovered, exposed to the air; hence this superficial necrosis is rarely a process of decomposition, seldom accompanied by bad smells. In deeply-situated necrosis, as in that of a whole diaphysis or of a sawed or fractured surface, which is embedded in soft parts, there is usually decomposition of the medulla; the smell from a large extracted sequestrum is occasionally very penetrating. This decomposing medullary substance is dangerous as long as no line of demarcation has formed, while the lymphatic vessels of the vicinity are still open; when the proliferation of tissue has occurred in the borders of the bone next the healthy parts, the inflammatory neoplasia forms a wall through which reabsorption does not readily occur. How are we to recognize a deeply-situated sequestrum? This can only be exactly done by the probe. Through the opening from which the pus flows we pass a probe, as large a one as possible, with which we feel the surface of the sequestrum, which is usually smooth and firm, more rarely rough and soft. We attempt to slide the probe along it, to determine the length of the sequestrum; we also press the probe firmly against the sequestrum, to find whether it be movable, detached, or whether it be still firm; as you will understand, this is important in relation to the question whether we may as yet attempt extraction of the sequestrum. A further aid to diagnosis is the increased thickness of the extremity; we feel the extensive new formation of bone; thick yellow, often mucous, pus flows from the openings; the bone is not especially sensitive to pressure; nor is careful probing usually painful, although the patient often dreads it, because some surgeons do it with unnecessary violence, but without any result. The patient is free from fever.

From these points you will readily diagnose many cases of necrosis; as long as there are no external openings, the diagnosis of central necrosis of a bone is liable to error. Caries is almost the only thing for which necrosis can be mistaken; the mode of origin and the locality aid greatly in the distinction, for necrosis occurs more frequently as a result of acute inflammation in the hollow bones (*femur*, *tibia*, *humerus*), caries usually occurring more slowly in spongy bones; however, the objective symptoms are also different: in caries there is but little formation of new bone about the ulcer, often none can be felt; in necrosis this is extensive: in caries the pus is thin, bad, serous; in necrosis it is thick, often good, frequently mucous: in caries we pass the probe into rotten bone, and probing is usually quite painful; in necrosis the probe generally strikes on the firm sequestrum and is not often painful. From this comparison of the symptoms, which result from the different natures of the two diseases, you must acknowledge the possibility of a diagnosis; in many cases, indeed, it is very easy and simple. In other cases, the anatomical conditions are more difficult to understand; when necrosis and caries occur together, all the symptoms, except feeling the sequestrum on probing, are in favor of caries. In central caries of the hollow bones, enormous thickening of the bone occurs in exceptional cases, at the same time the inner wall of the bone-cavity may feel very firm and hard, like a sequestrum; these cases may give rise to error: on opening the cavity, no sequestrum is found, as had been expected; it is possible that in these rare cases the sequestrum may have been very small and may have been absorbed; of this more hereafter. But these exceptional cases do not disprove the rule; hence you may, to a great extent, confide in the above comparative diagnosis.

Now, a few words about the fate of the sequestrum. Do you mean to say the dead bone cannot be reabsorbed? Have I not told you frequently that dead bone may be dissolved and consumed by the granulations? Hence we should expect that the elimination of the sequestrum would not require any aid. From my observations, I have no doubt that small sequestra may be completely consumed by proliferating granulations; granulations that are being destroyed or undergoing cheesy degeneration have no power of dissolving bone; we have already stated, when speaking of caries, that partial necrosis occurs so readily in atonic suppurative or caseous ostitis, just because the inflammatory neoplasia, which so quickly breaks down again, does not dissolve the bone, but leaves it to be macerated in the body. But the reabsorption of the sequestrum has its limits: first, of course, it ceases where the bone is uncovered, for here the granulations have no effect; it also ceases as soon as they secrete pus on their surface; hence a sequestrum, resulting from acute periostitis, is not usually absorbed at the point where the periosteum suppurates and where pus forms during the whole process, because it does not come in contact with the granulations; but at all points where the sequestrum must be loosened, reabsorption commences from the interstitial granulation-masses forming on the bone; lastly, after the sequestrum is detached, if these granulations also produce pus, reabsorption ceases here also, and the sequestrum bathed in pus ceases to decrease; the granulations of the pus-cavity, growing from all sides toward the sequestrum, in the course of time undergo chemical change; they become very gelatinous, mucous, and often undergo fatty degeneration.

But the sequestrum must finally come out. Can it do so unaided? This does occur; whence the power that pushes it out? Let us suppose a central necrosis; a sequestrum becomes detached from all sides; then, for the reasons above mentioned, it is considerably smaller than the cavity in which it lies; the piece of bone is now quite loose; granulations grow toward it from all sides except from the one where the pus-cavity opens externally; here there is no resistance; if the opening be large enough, the constantly-increasing granulations push out the sequestrum. But for this to occur there must be certain mechanical conditions which are rarely fulfilled; small sequestra are often thrown off spontaneously; large ones, which cannot pass the existing openings, must be removed artificially.

The *treatment* of necrosis at first consists simply in keeping the fistulæ clean. Chemical solution of the sequestrum is not to be thought of. If you were daily to pour muriatic acid into the fistulous opening, it would affect the newly-formed osseous tissue as much as, or more than, it would the sequestrum, which would be very unfortunate, as it must replace the latter. Hence *the mechanical removal of the sequestrum* is the only thing left; *this should not be attempted before complete detachment.* This is a very important rule: first, because the dead bone can rarely be sawed out without removing a good deal of the healthy and of the newly-formed bone, both of which are bad; and, secondly, because the new bone is rarely firm enough before the sequestrum is detached. Here, again, we meet a wonderful provision of Nature: the sequestrum is not generally detached till the new formation of bone is strong enough to replace the lost portion of bone. This beneficent provision should not be brought to naught by meddlesome interference. There are only a few special exceptions to the above rule, especially in necrosis from phosphorus, which is not a pure necrosis, but is often combined with caries; but of this we shall treat more particularly in special surgery and in the clinic.

I have already told you that we may sometimes tell by the probe whether a sequestrum is detached; but this is not always so; it may be so shut in by granulations that it cannot be felt to move. It is always hard to decide on the mobility of a large sequestrum; and the curved shape of the bone (as of the lower jaw) may greatly interfere with the decision. In such doubtful cases the duration of the process, and the thickness of the bony case, are important aids in determining whether the sequestrum be detached or not. Most sequestra are usually detached in eight or ten months; in a year even an entire necrotic diaphysis usually lies as a loose sequestrum in the newly-formed bony case. These are approximate determinations, which may of course have exceptions. If the formation of bone be still weak, and nevertheless the sequestrum be already detached, it is well to postpone the extraction in the humerus, tibia, and femur, so that the formation of bone may be firmer, provided the general health does not suffer. Should albuminuria begin, the extraction should be hastened.

Extraction of the sequestrum, especially when it requires preliminary enlargement of the cloaca (fistulæ leading into the bony case), is called the *operation for necrosis or sequestrotomy.* This operation may be very simple. If one of the openings of the bony case is tolerably large, and the sequestrum small, we may pass a good pair of forceps through the opening and try to seize and remove the sequestrum. If, as in caries necrotica, there be no formation of new bone, we enlarge the fistulous opening through the soft parts with a knife, and remove the necrosed piece of bone. But, if the opening be small and the sequestrum large, a portion of the bony case must be removed, both for the purpose of introducing instruments for traction and for removing the sequestrum. In rare cases, it is sufficient to enlarge *one* opening with trepan, chisel, and hammer; I usually do the operation as follows: With a stout knife I make an incision through the soft parts down to the bony case, from one fistulous opening to an adjacent one; then, with a handled scraper, a *raspatorium*, I draw the thickened soft parts from the rough surface of the bony case, so as to expose it to a certain extent. This exposed portion should now be removed, to make an opening through which the sequestrum may be removed. For this purpose we may use saws of various kinds—the osteotome, the panel-saw, etc.; of late, I always employ chisel and hammer; the work is laborious, use what instruments we will. The portion of the bony case removed should be as small as possible, so as to interfere the less with its firmness. When the case is opened, the sequestrum is exposed; we attempt its removal by elevators or with strong forceps; this also is sometimes very troublesome. When the removal is accomplished, the indication is fulfilled.

If, contrary to expectation, the sequestrum be found not detached, we should avoid forcing it out, but wait a few weeks or months, till we are satisfied of its detachment. After the operation, the suppurating cavity in the bone is to be kept clean; the patient should keep his bed for some time; most fistulæ soon cease discharging, but it is still some time before the sequestrum-cavity is filled with ossifying granulations. We cannot do much to hasten this, and the fistulæ, which sometimes remain a long while, usually cause so little trouble that we are not often called on to do any more operations for them. Occasionally, however, too large an opening remains for a long time, its walls become sclerosed and cease to granulate; here we apply the treatment for atonic ulcers of the bone. In these old cases, the hot iron to the cavity in the bone, and chiselling out the track of the fistula, is the only treatment from which I have ever seen any benefit. Many cases of these bone-fistulæ are incurable.

The full value of sequestrotomy has only been appreciated for the past ten years; it first became common after the introduction of chloroform, for it is a terrifying operation. This chiselling, sawing, and hammering on the bones, are horrible for a looker-on, and the more so as the operation may last some time; amputation is a trifle in comparison. Local anæmia (as induced by *Esmarch's* bandage) greatly facilitates the recognition of the anatomical conditions in these operations. Formerly amputations were frequently performed for total necrosis, a thing that no surgeon would do now. Hence, in old museums, you find the most beautiful preparations of extensive necroses; now these are rarely found, because almost all sequestra are removed at the proper time. Locally the operation is quite extensive, but the febrile reaction is usually slight. Severe as the inflammatory symptoms and fever might be if you were to treat a healthy bone in the same way, the effect on the bony case of the sequestrum is but slight. From my own experience, I do not know of a case which, after such an operation, even where the entire bony case was opened in total necrosis of the tibia, turned out badly; and I am satisfied that the operation for necrosis is one of the most successful of operations, and that by it many lives are saved, such as were formerly lost from amputation, from constitutional diseases due to continued suppuration from the bone, or from fatty degeneration of internal organs, morbus Brightii, and tuberculosis.

## LECTURE XXXVI.

### APPENDIX TO CHAPTER XVI.

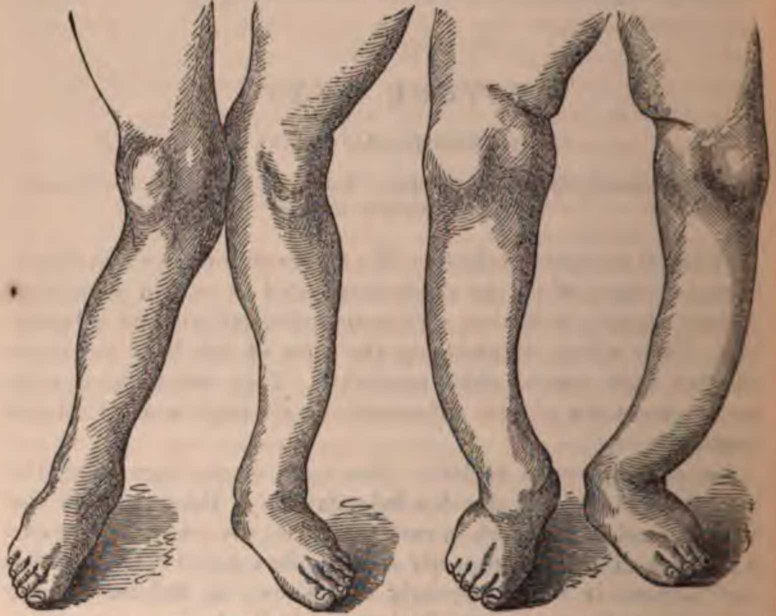
Rachitis: Anatomy, Symptoms, Etiology, Treatment.—Osteomalacia.—Hypertrophy and Atrophy of Bone.

*Rachitis and Osteomalacia.*—We must still touch on two constitutional diseases, which are chiefly manifested in certain changes of the bone, namely, softening. They are called rachitis and osteomalacia. Their effects in changing the form of the bone are much alike, but their natures differ somewhat. They cannot be exactly classed among the chronic inflammations, although nearest related to this process.

Let us begin with rachitis. The name comes from ῥάχις, the backbone, and properly signifies inflammation of the spine; but the vertebræ rarely suffer much in rachitis; hence the origin of the name is not very clear; subsequently it was often called "English disease," because it was particularly well known to English writers, and probably also was especially frequent in England.

The essence of the disease consists in deficient deposit of chalky salts in the growing bone, and remarkable thickness of the epiphyseal cartilages. You will already see that this disease is peculiar to childhood ; it is a disease of the development of bone, which h[illegible] ever usually affects so many bones that it must be regarded, no[illegible] a local, but as a constitutional disease, which you may reckon am[illegible] the dyscrasiæ already known to you. The insufficient deposi[illegible] chalky salts in the growing skeleton in rachitis is accompanied b[illegible]y normal development of vessels and increased absorption of the [illegible] bony tissue already developed (during the growth of bone there is always a slight amount of absorption at the inner and outer surface of the cortical layer), as well as unusual proliferation of the epiphyseal cartilages ; if you remember also the young osteophytes forming on the outsides of the hollow bones, it must be acknowledged that this disturbance of nutrition can scarcely be distinguished from inflammation, even if it passes on to suppuration and caseous degeneration. We often find rachitic symptoms in scrofulous children, and some physicians regard the disease as one symptom of scrofula ; but this is not quite correct, for in many rachitic children we find no traces of scrofula, among which are especially reckoned tendency to swelling of lymphatic glands, suppuration, and caseous degeneration. Moreover, the rachitic process has little anatomical connection with

FIG. 91.



Typical illustrations of rachitic malformations of the leg.

the forms of periostitis and ostitis that we have studied in scrofulous children, for it never leads to caries. The disproportion between the growth of the bone and deficient impregnation of its tissue with chalky salts results in lack of firmness of the bones; consequently they bend, especially those that bear the weight of the body. Where the bones are very soft, muscular contraction also acts on them so as to induce curvature. These curvatures are most common in the lower extremities; the femur bends anteriorly and inwardly, the bones of the leg bend anteriorly and outwardly or inward. The thorax is compressed laterally so that the sternum projects sharply, and the result is the so-called chicken-breast (*pectus carinatum*). In high grades of rachitis there are also distortions of the pelvis, spinal column, and upper extremities. In such children the occiput long remains soft and compressible, and dentition is delayed. Sometimes the softness of the occiput is the sole symptom of rachitis, so that this has even been regarded as independent of the general rachitic disturbance. According to *Virchow*, the distortion of the upper extremities depends mostly on a number of small curvatures (infractions) of the entire bone, or of parts of the cortical layer. Complete fractures rarely occur; if they do, the bone is again united firmly by callus, under the ordinary treatment.

Rachitis causes other changes in the bone besides these deformities, namely, thickening of the epiphyses and of the point of union between the costal cartilages and the bony ribs. The thickening of the epiphyses may be so great, at the lower end of the radius, for instance, that above the wrist, at the point just above the epiphyseal cartilage, there is a second depression in the skin; this appearance of the joint has given rise to the term "double-jointed;" the nodular thickenings on the anterior ends of the ribs are often very remarkable, and, as they lie regularly under one another, they have been called the "rachitic rose-garland." If these changes in the bone have taken place, there is no hesitation in diagnosing rachitis; before they have become evident, the diagnosis is doubtful. It is true, there are some prodromal symptoms: voracious appetite, pot-belly, disinclination to standing and walking; but these symptoms are always too undecided to permit any definite conclusion. The disease most frequently begins in the second year, and attacks well-nourished or even fat children; indigestion and inclination to constipation occur occasionally, but not always. We know little of the exciting causes of rachitis; here in Germany it is about equally frequent in all classes of society; hereditary influence may have some effect; we may suspect, but cannot prove, a disturbance in the composition of the blood, in the assimilation of nutriment. In regard to the course of the disease, under proper treatment it often subsides quickly; that is, symptoms of distortion of the bone cease, or rather do not increase; the children, who had ceased to walk, again desire to do so. As the normal growth of the bone goes on, the distortions become less perceptible, and often disappear entirely; this may be readily understood from the nature of the growth of the bone. Before the bones again acquire their normal consistence, at the end of the rachitic process, there is usually an abnormally rich deposit of bone, so that in certain stages the rachitic bones are abnormally hard and firm; that is, in a sclerosed state. Rarely, rachitis lasts till the skeleton has attained its growth, and these cases furnish the excessive distortions and dislocations that are usually presented as types of this disease. In every pathological anatomical collection you find examples of such rachitic skeletons.

The greater my experience, the more I am inclined to regard *flat foot*, *genu valgum* and *varum*, as well as *lateral curvatures* of the spine (scoliosis), as being due to weakness of the bones, which cannot be distinguished from a mild form of rachitis. This localized rachitis comes later in life, it is true, but it is generally between ten and twenty years, while the disease briefly termed rachitis, as above stated, is mostly seen in very young children; still both cases are due to the bones remaining soft and to pliability of growing bones, besides which various other causes must act to induce the above-mentioned distortions.

Hereafter you will often hear that some physicians think there is a direct relation between rachitis and infantile diseases of the brain, especially paralyses, spasms, and psychical disorders. I will not deny that this rather obscure disease may directly affect the development of the brain, but in most cases it does so indirectly. The rachitic process in the cranial bones is often followed by rapid sclerosis, by such formation of new bone that even the cranial sutures may ossify; this interferes with the regular growth of the skull, which becomes irregular, and here and there too small for the growing brain, and thence arise functional disturbances of the brain.

Rachitic children are rarely brought to the doctor before the parents notice the thick limbs or distortion, or until, as the mother expresses it, "they are off their legs," i. e., they no longer wish to stand or walk, as they formerly did; the disease is so common and so well known that often it needs no surgeon for its recognition. As a rule, *treatment* has only one indication, that is, to remove the diathesis; hence it is chiefly medical, and especially dietetic. Regarding the latter, the patient should avoid too free use of bread, potatoes, mush, and flatulent vegetables; he should freely consume milk, eggs, meat, and good white bread, and should take strengthening baths of malt, herbs, etc. Internally we should prescribe cod-liver oil, iron, and similar strengthening and tonic remedies. We might think of giving preparations of lime, but they are so indigestible, and are so quickly excreted by the urine, that they do no good: they have almost been thrown aside; it is possible, also, that rachitis is essentially a disease of digestion, in which the preparations of lime are, from some unknown cause, not absorbed. It is rather a one-sided view to suppose that in rachitis or osteomalacia lack of supply of lime is the cause of absence of deposit of chalky salts in the bones, of the disappearance of that which has been deposited. It is also possible that lime entering the stomach, from faulty digestion, does not reach the blood, or that it is excessively excreted by the kidneys, or perhaps the newly-formed bony tissue does not take up the chalky salts brought to it in normal or even in excessive amounts. It is true, these points furnish no direct indications for treatment, but I mention them so that you may see that we are not physiologically justified in referring the disturbed nutrition solely to deficient supply. Frequently the parents ask for splints to remove the curvatures, or, at least, prevent their increase; they will also ask you whether the children should be urged to walk, or permitted to lie still. On this point it is best to let children have their own way: if they do not wish to go, do not urge it; if they lie still more than they run about, they should be kept in the open air as much as possible; taking children from a damp city dwelling to the country often suffices for the cure of rachitis. Splint boots and similar apparatuses, that load the feet, should only be applied in cases of excessive curvature, where the position of the feet mechanically interferes with walking; this state of affairs is rare, hence the indication for such orthopedic apparatus is limited.

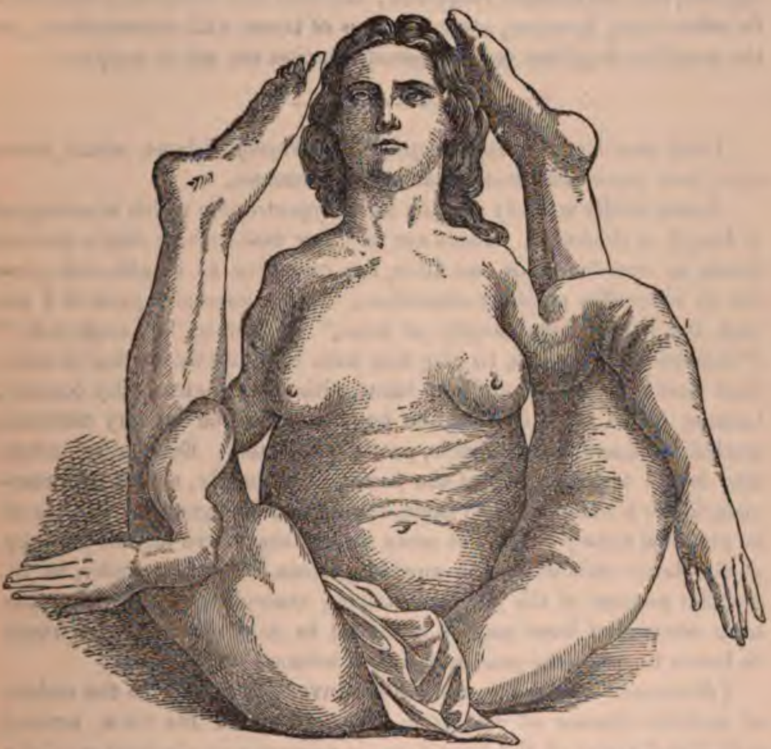
When the rachitis has disappeared, such amount of curvature may remain in rare cases as to require some treatment; in the great majority of cases this is unnecessary, since, as already stated, the curvatures spontaneously disappear with the growth of the skeleton. Only in the leg curvatures sometimes remain, so that the foot is distorted, and only its inner or outer border can be placed on the floor; if this remains for years at the same point, an attempt should be made at straightening. This may be done in two ways. We anæsthetize the child, and carefully fracture the bone subcutaneously; have the leg held straight, apply a plaster-bandage, and treat the injury as a simple fracture; recovery usually takes place readily. In some cases, however, after the rachitis has run its course, the bone is so very firm that this breaking does not succeed. Then subcutaneous osteotomy, according to *B. von Langenbeck* (p. 230), is indicated. The results of this operation, which I have had to make four times, have so far been very satisfactory; in one of these cases the skin-wound healed by first intention, and the subsequent treatment was that of simple fracture. The operation will always remain a rare one, because these excessive rachitic distortions are themselves rare.

Now, a few words about *osteomalacia*, bone-softening, *κατ' ἐξοχήν*. The disease only occurs in adults, and is also characterized by distortion of the bones; but here there is an actual reabsorption of existing bone. In the hollow bones the medulla gradually assumes the preponderance, while the cortical substance becomes thinner and thinner, and consequently the bones weaker and more flexible; and finally there may be a complete absorption of the bone, so that little is left besides the periosteum, which participates rarely, and then but little, in the disease, scanty osteophytes growing from it. The spongy bones also grow weaker, the trabeculæ thinner, and become so soft that they shrink. The medulla appears reddish and gelatinous, but does not, as in fungous caries, consist solely of granulations; it contains much fat. The microscopic appearances in this process have already been described in ostitis malacissans. Lactic acid has been found in the medulla of the hollow bones, so that it is very probable that the bones are dissolved by it. The lime going into the blood is often excreted in the urine as oxalate of lime. So you see that this is an ostitis malacissans with nothing peculiar in its anatomy, but which owes its distinction to its affecting many bones simultaneously, often occurring under peculiar conditions, and never leading to suppuration or caseous degeneration.

Concerning the etiology of the disease we know but little; osteomalacia is particularly frequent in some parts of Europe, and among women; it attacks the latter more particularly while in the puerperal condition; occasionally it is preceded by drawing pains and soreness on moving, which continue through the disease. The distortions occur chiefly, primarily, even solely, in the pelvis, which assumes a peculiar, laterally-compressed form, of which you will hear more in obstetrics. This is followed by curvature of the spine and lower extremities, with muscular contractions. The disease may pause, and exacerbate with a new pregnancy, etc. Slight grades and localized forms of osteomalacia, as that of the pelvis, not unfrequently recover spontaneously; if the disease be of a high grade, general marasmus occurs, and the patient dies. The treatment is the same as in rachitis, but the hopes of success are less.

The cases of local osteomalacia or osteoporosis, which often accompany caries, are more interesting to us than the above-described general osteomalacia. I will relate you a case that will at once explain what I mean: A woman, about forty years old, was brought to the hospital for extensive caries of the knee-joint; she was excessively marasmic, and died the following day. On autopsy we found complete fatty degeneration of the liver, spleen, and kidneys; in the knee the condyles of the femur and tibia were extensively destroyed by the carious process. I sawed off the lower end of the femur to remove the preparation, and found that it was very much thickened; the cortical layer measured scarcely half a line; the medulla was reddened, and resembled that in osteomalacia; the thinning extended upward to the trochanter. I examined the tibia of the diseased leg, the femur of the healthy one, and the pelvis, and found them all perfectly normal; that is, only the femur of the diseased leg was osteomalacic. In the

FIG. 92.



Woman with excessive osteomalacia, after *Morand*. The bones consist mostly of membranous cylinders, or very thin layers of bone.

same way I once found the lower half of the tibia affected with osteomalacia, in caries of the ankle. There was apparently the same thing in a child that had the head of one femur removed for caries of the hip-joint. I assisted in this operation; as I was on the point of lifting the thigh and rotating it outward to aid the operator, the thigh broke through the middle, right in my hands; a plaster-bandage was applied, and the fracture recovered; the child was completely restored. In other cases, however, after fractures of bones with osteomalacia, in the so-called fragilitas ossium, pseudarthroses are apt to remain.

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I will also mention *hypertrophy* and *atrophy* of bone, which, however, have more anatomical than clinical interest.

Anatomically we may call any bone hypertrophic which is enlarged in length or thickness. There are very few cases where single hollow bones, as one femur or one tibia, are excessive in length, and give rise to inequality of the extremities; for this excessive growth I accept the name "hypertrophy of bone," or, better, "giant-growth" ("riesenwuchs"); still, to give this term to every thickening or sclerosis would be of no practical value, although anatomically correct, because these conditions of the bone may depend on very different morbid processes, partly active, partly completed. Even more indefinite is the term *atrophy* of the bone; occasionally, a carious, osteomalacial, or a half-destroyed bone, etc., is thus designated. This is of no practical value; we do not mean to deny that there may be atrophy of the bone without a true morbid process. Senile atrophy, as of alveolar process of the jaw, is a striking example of this. Here the term atrophy of bone may be retained; in most other cases it would be better to name the process that has induced the atrophy.

[*Kassowitz* has lately made some investigations as to the nature of rachitic disease of the bone. According to his view, around each blood-vessel of the bone there is normally an absorption of the bone caused by currents of fluid, and probably effected by the free carbonic acid of the blood; and that, in rachitis, chronic hyperæmia and morbidly-increased new formation of vessels in the ossifying cartilage and in the periosteum as well as in the growing bone, are the causes of the hinderance of the deposit of chalky salts and of the increased absorption of those already deposited. The holes left by absorption of the bone fill with young bony tissue; but this does not ossify normally. He thus explains the irregular form of the bony lamellæ, and of the surface of the bone as found in rachitic skeletons.

*Rokitansky* had already asserted that rachitis was connected with vascular development, and, from recent knowledge of the power of carbonic acid in dissolving bone, it is probable that the softening is due to the vascularity. Investigations about the growth of bone, ossification, and reabsorption, are so difficult that it need not astonish you that there is no universally accepted theory on this point.]

# CHAPTER XVII.

## *CHRONIC INFLAMMATION OF THE JOINTS.*

### LECTURE XXXVII.

General Remarks on the Distinguishing Characteristics of the Chief Forms.—*A.* Fungous and Suppurative Articular Inflammations (Tumor Albus), Symptoms, Anatomy, Caries Sicca, Suppuration, Atonic Forms.—Etiology.—Course and Prognosis.

In more than half the cases of chronic inflammation of the joints, the synovial membrane is the part first affected; this affection may be accompanied by more or less secretion of fluid, and this fluid may be purely serous or purulent. Chronic serous synovitis (*hydrops articulorum chronicus*), unless from some external cause, is no more apt to become purulent synovitis than is chronic articular rheumatism, But other forms of chronic inflammation of the joints may be accompanied by suppuration from the first, or else may be characterized by the formation of numerous granulations. The two chief groups of chronic articular inflammation are characterized by the condition of the synovial membrane even more than by the quality of the fluid contained in the joint; when the secretion is purely serous, the synovial membrane is somewhat thickened, it is true; the tufts are enlarged, and their apices are somewhat more vascular than normal, still these changes are never so extensive as to greatly injure the membrane; but in the other variety of chronic inflammation the membrane changes greatly, and is gradually transformed into a spongy (fungous) mass of granulations, which often, but not always, produces pus, opens outwardly (fistula, cold abscess), causes distortion of the cartilages and bones, and may thus induce peripheral caries of the epiphysis. This latter group, which has several subvarieties, we shall term *fungous* and *suppurative inflammations of the joints;* they form the great majority of all articular inflammations, and hence will occupy our attention for some time. For a more exhaustive account of joint diseases, I refer you especially to the excellent works of *Bonnet*, *Volkmann*, and *Hueter*.

### A THE FUNGOUS AND SUPPURATIVE ARTICULAR INFLAMMATIONS. (TUMOR ALBUS).

Tumor albus (white swelling) is an old name which was formerly applied to almost all swellings of the joints that ran their course without redness of the skin; now it has been agreed only to give this name to the affection we are about to describe, which is also, with more or less correctness, termed *scrofulous inflammation of the joint;* but of this later.

The disease is very frequent in childhood, particularly in the hip and knee joints; it usually begins very insidiously, more rarely subacutely. If the knee-joint be affected, the parents usually first notice a slight dragging or limping of the lame leg; the child, either voluntarily or on questioning, complains of pain after walking some distance, and on pressure over the joint; about the knee itself the laity can see nothing out of the way. On comparing both knees, the surgeon will find, even quite early in the disease, that the two furrows which normally run alongside of the patella, when the limb is extended, and give the knee-joint its shapeliness, have either disappeared on the affected side or at least are shallower than on the sound side; except this there is nothing observable. The hinderance to walking is so slight that children go about with a slight limp for months, and complain so little that it is some time before the parents feel obliged to consult a surgeon; they often delay doing this till, after continued exertion, the limb has begun to pain and swell more. The swelling, which was at first scarcely perceptible, is now quite evident; the knee-joint appears evenly round and quite sensitive to pressure. If we suppose that no treatment be instituted, but the disease left to itself, its course is about as follows: The patient continues to limp around for a few months, but finally the time comes when he cannot walk; he is obliged to lie down most of the time, because the joint is so painful; gradually it becomes more and more angular, especially after each subacute exacerbation. Now, certain parts of the joint, at the inner or outer side, or in the hollow of the knee, become more painful; there is evident fluctuation at some one of these points; the skin grows red, and finally suppurates from within outward, and is perforated after a few months; a thin pus, mixed with fibrinous cheesy flocculi, escapes. Now the pain decreases, the condition improves; but this improvement does not last long; a new abscess soon forms, and so it goes on. Meantime, perhaps two or three years have elapsed, the general health of the patient has suffered; the child, which was previously strong and healthy, is now pale and thin; the opening of the abscesses is not unfrequently accompanied or followed by fever; this fever exacerbates as each new abscess develops; this exhausts the patient; he loses his appetite, digestion is impaired, diarrhœa comes on, and the emaciation is increased from week to week. Even at this period the disease may spontaneously subside, although this rarely happens; more frequently it proves fatal, from the exhaustion caused by the suppuration and continued hectic fever. Should recovery take place, it is announced by decrease of the suppuration, retraction of the fistulous openings, improvement of the general health, increased appetite, etc.; finally, the fistulæ heal, the joint remains angular or distorted in some way, the pain ceases, and the patient escapes with his life and a stiff leg; this termination of chronic suppuration of the joint in *anchylosis* (stiff-joint) is the most favorable that can occur when the disease has been severe; the anchylosis may be complete or imperfect, i. e., the joint may be perfectly stiff or slightly movable; the whole process may have lasted from two to four years.

Among the local symptoms I may add that for each joint certain muscles gradually become permanently contracted; usually it is the flexors; in the hip-joint the adductors and rotators are also affected, and the joint is permanently held in such a position as to give little or no pain. If these pathological positions are caused by the muscular contraction alone, and have not continued too long, they may at once be overcome by anæsthesia. But after months or years atrophy begins in the fasciæ, and afterward in the muscles, which even under anæsthetics can only be broken up by force. After long disuse the muscles are greatly atrophied by fatty degeneration and cicatricial contraction. The articular capsule too, which was much infiltrated and swollen, as well as the accessory ligaments, also contract on the side toward which the joint has been bent; thus, in the case of the knee-joint this contraction would be greatest in the hollow of the knee.

Cases where the disease begins with a seropurulent effusion in the joint (catarrhal, blennorrhœal synovitis) are rare. I have seen them chiefly in tuberculous patients. The symptoms are at first the same as in chronic dropsy of the joint, but the joint is painful and its function more impaired. Comparatively often ostitis and periostitis near a joint are the causes of synovitis; one or other side of the condyles of the femur, tibia, or humerus, or the posterior surface of the olecranon, become painful; the pain remains localized for some time at one point; then there is a doughy œdema, and finally an abscess. Meantime the functions of the joint may remain unimpaired for months, till the suppuration (occasionally with acute inflammatory symptoms) attacks the joints; then the course above described begins. In some cases these abscesses always remain periarticular, and heal before the joint is opened; this might cause peri-articular cicatricial contraction, while the joint was perfectly healthy.

["As the disorder advances, the alterations become more distinctly defined, for, as the disorganizing process is now in full play, its devastating effects are plainly visible in every portion of the joint. The lymph increases in quantity, and is often intermixed with sero-purulent matter, or thick, greenish-looking pus. The tufts of the synovial membrane are converted into soft, gelatinous, fungous granulations, which gradually cover the cartilage, extend into and finally destroy it, imparting to it an ulcerated or worm-eaten appearance. The bony structure is finally invaded by the granulations, rendering it very red, soft, carious, rough, and easily crumbled. The ligaments exhibit well-marked signs of inflammation, being loose and spongy at one point, attenuated at another, and perhaps thickened or hypertrophied at a third."]

Lastly, the bones may be primarily attacked by ostitis malacissans; this occurs particularly in the carpus, tarsus, and caput femoris of feeble patients; the joints may long remain unaffected, even if periosteal abscesses with great œdema and suppurating fistulæ form. In primary diseases outside of the epiphyses muscular contractions are less apt to develop than in primary disease of the synovial membrane and primary subchondral ostitis.

This short description may serve you for a general type of the disease in question, and of its importance; to enable you to understand the various forms in which it may appear, it seems advisable to first give you a clear description of the anatomical changes in these diseases of the joint. We have the opportunity of observing the different stages of these changes in exsected joints, in amputated limbs, and on the dead body. I have paid so much attention to this subject, that from my individual observations I can give you a very accurate account of the anatomical changes. These are much alike in all cases, and, from what you already know about chronic inflammations of other parts, you will anticipate that there is in reality only a variation of the old story of serous and plastic infiltration with various grades of vascularization, of proliferation, and destruction, etc.

Let us first with the naked eye study these joints in various stages of the disease. Let us suppose the common case of the affection beginning with chronic synovitis: we first find swelling and redness of the synovial membrane; it has already undergone some change in the lateral portions of the joint, in the folds, and neighboring sacs; its tufts are puffed up, very little elongated, but very soft and succulent; the whole membrane is more readily distinguished from the firm tissues of the capsule, and may be detached with greater facility than normal ly. At this time the synovia is rarely increased, but is cloudy, or even resembles muco-pus. These changes in the synovial membrane gradually increase; it becomes thicker, more œdematous, softer, redder; the tufts grow to thick pads, and in places resemble spongy granulations. The surface of the cartilage loses its blue lustre, though it is not yet visibly diseased; but the synovial outgrowths begin to grow over the cartilages from the sides, and to push in between the two adjacent surfaces of cartilage; meantime the capsule of the joint is also thickened, and has acquired an evenly, fatty appearance, and is very œdematous; this swelling and œdema gradually extend to the subcutaneous tissue, and to the skin. From this point, the changes in the cartilage claim most of our attention. The synovial proliferations, in the shape of red granular masses, advance gradually over the entire surface of the cartilage, and cover it completely, lying over it like a veil (Fig. 93);

FIG. 93.

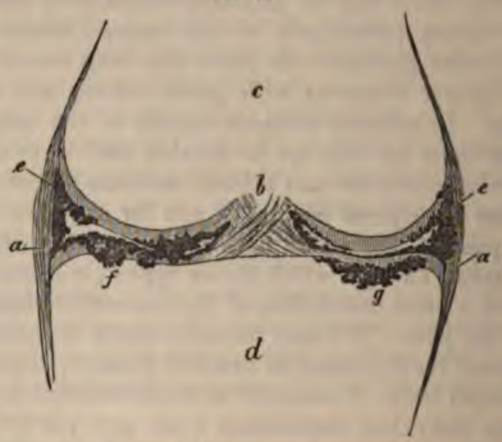


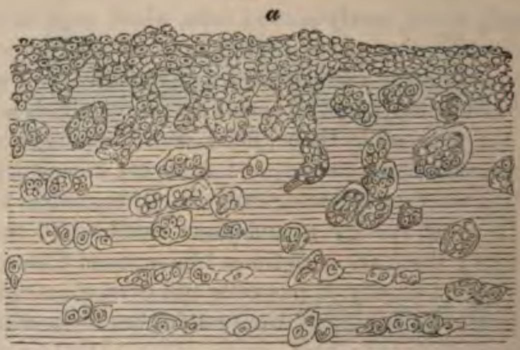
Diagram of a section of a knee-joint (the interarticular cartilages have been left out, the articular cartilages shaded) with fungous inflammation: *a a*, fibrous capsule; *b*, crucial ligament; *c*, femur; *d*, tibia; *e e*, fungous synovial membrane growing into the cartilage, at *f* it even grows into the bone; at *g* are isolated prolifications of the granulations into the bone on the border between bone and cartilage.

if we attempt to remove this veil, we find that in some places it is attached quite firmly by processes entering the cartilage, just as the roots of an ivy-vine cling to and insert themselves into the wall against which it grows (as is also the case in pannus of the cornea); these roots not only elongate, they spread out, and gradually eat up the cartilage, which, when the covering of fungous prolifications is removed, appear first rough here and there, then perforated, and finally disappear altogether; then the fungous prolification extends into the bone, and commences to consume this; the result is fungous caries, as we have already learned; as a result of the changes from chronic inflammation, the bone is destroyed in the manner before described, and here you have the whole course and the relation of fungous inflammation of the joint to caries. The morbid process advances unequally; one condyle of a joint may be almost consumed while another partly preserves its cartilaginous surface. The other parts of the synovial membrane may also proliferate outwardly toward the capsule; capsule, subcellular tissue, and skin, are transformed at one place or another into fungous granulations, with or without suppuration, and thus we have external openings, and fistulæ, which either communicate directly with the joint, or with a synovial sac.

Here let us stop a moment to notice what may be seen with the microscope at the affected part; on this point I can give you least that is new. The normal synovial membrane consists of loose connective tissue with moderately rich capillary net-work, which forms complicated folds in the tufts; on the surface of the membrane there is a simple layer of endothelium, composed of flat polygonal cells, just as there is on most serous membranes. The tissue of the membrane is gradually permeated with cells, becomes softer, loses its firm, fibrous character, and the vessels dilate and increase decidedly. The endothelium is destroyed in limited layers of flat scales; its place is supplied by small, round, newly-formed cells, which soon unite with the constantly-degenerating tissue of the synovial membrane, and cease to be distinguishable as separate layers. Through the progress of the plastic infiltration the synovial membrane gradually loses its former structure; the connective tissue, filled with innumerable new cells, gradually becomes homogeneous, and from the constantly-increasing vascularization the tissue histologically exactly resembles that of granulations. In these spongy granulations small white nodules form here and there; these are sometimes like mucous tissue, sometimes they are composed chiefly of pus-cells and even giant-cells.

[The tuberculous nature of the nodules found in white swelling has long been disputed: here, as in other pathological products whose origin is doubtful, inoculation has been tried; and *König*, by inoculating some of the fungous proliferations of joints, has induced miliary tuberculosis, while *Hueter* introduced isolated nodules from a synovitis fungosa into the anterior chamber of the eye of a rabbit, and this was followed by choroidal tubercle, and after a long period of incubation by general tuberculosis. These experiments have been repeated by many pathologists and surgeons, and there can be no doubt that most cases of so-called granular or fungous synovitis are of tubercular nature. On the other hand, it is certain that in some persons, at least, tuberculosis of the joints is a primary disease, and for a long time remains local, so that on autopsy the only tuberculous point found is in the joint. The question, "How does the tuberculous virus reach the synovial membrane?" is just as difficult of solution as in the case of primary tuberculosis of the bones. It has long been known that injuries of the joints, especially contusions, sprains, intra-articular hæmorrhages, etc., often excite the disease; but this does not explain how these subcutaneous injuries, to which the extravasation is the only thing in common, should cause the entrance of tuberculous virus. *Cohnheim* thinks that in these cases also the infection comes through the lungs or intestines, and then the virus circulates in the blood, perhaps with the corpuscles, and escapes at the point of injury through the vessels which have become permeable from the traumatic inflammation, or in the blood which has been extravasated into the tissue. *M. Schüller's* experiments have shown the possibility of inducing fungous inflammation of the joints by very slight injuries in animals that have been made tuberculous by inhalations. The significance of fungous synovitis, ostitis, periostitis, etc., termed local tuberculosis, is that at any time they may induce general tuberculosis.]

FIG. 94.

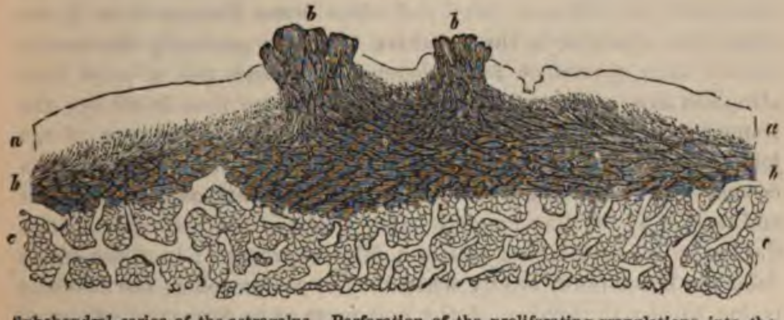


Degeneration of the cartilage in fungous inflammation of the joint. *a*, Granulation-tissue on the surface, magnified 350 diameters; after *O. Weber.*

But in cartilage there are also occasionally appearances which show that sometimes the cartilage-cells participate little in the cell-proliferation, so that there may also be a more passive absorption of the cartilage-substance from proliferation of the synovial membrane. The histological changes in the articular capsule and ligaments consist in serous and plastic infiltration which only attain a high grade at certain points, but generally only induce connective-tissue neoplasiæ, which to the naked eye resemble fatty thickenings.

Now that you have a general view of the anatomical changes in fungous inflammation of the joints, we may go more minutely into the various modifications; in so doing we shall start from the above-described course. So far I have represented the course of the disease as it occurs when originating in the synovial membrane, but there are also other starting-points for the disease; there may be a central, or more rarely a peripheral, caries in the spongy epiphysis of a hollow bone, or in one of the spongy bones of the wrist or ankle, and this may perforate from within outwardly through the cartilage, and thus excite synovitis. It also happens that, sometimes, along with the fungous proliferation of the synovial membrane, there is an independent proliferation under the cartilage, in the boundary between it and the bone (Fig. 93, *g*), which subsequently unites with that from above, so that the cartilage lies partly movable between the two granular layers. This occurs quite frequently, especially in the hip, elbow, and ankles. The cartilage is so loosened by this primary ostitis of the ends of the bone or sub-chondral caries, that it may be removed apparently intact from the subjacent, vascular, soft bone. It has already been mentioned that inflammation of a joint may be induced by acute periostitis and osteomyelitis; the inflammation then extends from the periosteum to the capsule of the joint, and thence to the synovial membrane; the anatomical changes are as above described. The infiltrations which we so often find around the sheaths of the tendons on the dorsum of the foot are often independent diseases of the cellular tissue of the periosteum and sheath of the tendon, but frequently they are due to ostitis of the ankle-bones. When an acute traumatic inflammation of a joint or an idiopathic acute suppurative synovitis passes into the chronic stage,

Fig. 95.



Subchondral caries of the astragalus. Perforation of the proliferating granulations into the joint: magnified twenty diameters: *a*, cartilage; *b*, granulations; *c*, normal bone, with medulla.

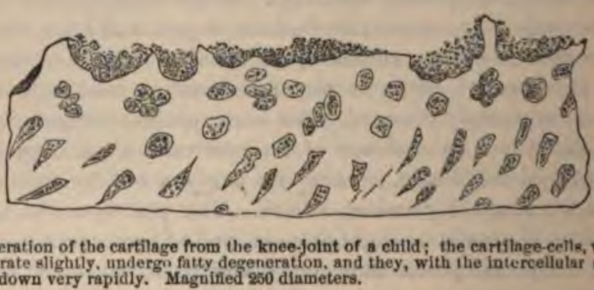
the same anatomical changes go on as in fungous inflammation. Chronic periostitis in the vicinity of the joint may also cause inflammation of the joint, especially when it induces cold abscesses; as may also chronic granular proliferations in the capsule, remains of neglected sprains of the joint.

The external appearance especially is greatly influenced by the extent to which the parts immediately around the joint participate in the inflammation; if the capsule participate very actively, the joint becomes regularly thick and round. This enlargement of the joint is also considerably increased by the formation of osteophytes, which form on the articular surfaces; these will be the larger, the more the capsule and periosteum of the articular surfaces have been implicated, and the more proliferating and productive the disease generally; while from the joint the condyles and sesamoid bones are destroyed, from without new bone is formed as described to you under caries. Caries of the joint has an old name, which is still occasionally used, it is *arthrocace;* this word is combined with the name of the different joints, and thus we speak of gonarthrocace, coxarthrocace, omarthrocace, etc. *Rust* wrote a book about diseases of the joint, and gave it the fearful name "arthrocacologie," which it is not worth your while to remember; I only mention it as a curiosity; it originated at a time when the study of eye-diseases also consisted almost exclusively in learning by heart the most frightful Greek names. The extent to which the muscles suffer in tumor albus is important. In the vicinity of the inflamed joint, and often some distance from it, the contractile substance in the primitive filaments gradually disappears, usually after precedent fatty degeneration, and the affected limb atrophies more and more, in some patients more than in others; the thinner it becomes, the more striking grows the enlargement of the joint, which often is not really very decided when you compare its measurement with that of the sound one. You will occasionally hear and read of the puffing up and enlargement of the articular ends of the bones in tumor albus; this is a false expression; in caries of the joint the bones never swell; when they appear swollen, the swelling is due to the thickening of the soft parts or to formation of osteophytes.

A further difference in the course of diseases of the joints lies in the greater or less tendency to suppuration; abscesses and fistulæ are by no means necessary sequelæ of fungous inflammations of the joints, they are rather accidents. You already know that caries fungosa not unfrequently runs its course without suppuration. The fungous articular inflammations are often accompanied by caries sicca; the affection may go on for years without the formation of abscesses, especially in adults otherwise healthy; there may be extensive destruction of the cartilages and bones, with the consecutive dislocations already mentioned under caries, without a drop of pus. If, in such a case of so-called caries sicca, you examine the granulations in the joint and bone, you will find them firmer than usual, and occasionally of almost cartilaginous consistence, like granulations that are about to atrophy or cicatrize; indeed, they do partly atrophy, but the proliferation often goes on again, and the bone is destroyed; the process is thus analogous to cirrhosis. Hence suppuration is by no means a measure for the extension of the process in the bone; on the contrary, the more luxurious the proliferation of the granulations, the more extensive the destruction. The dislocation of the bones, the deformity of the joint, is the most important measure of the extent of the changes in the bones and ligaments; if in a case of diseased knee the leg begins to rotate outwardly, and the tibia to shove backward, there is certainly destruction of part of the bone, and of a large part of the ligaments of the joint. In many cases fungous inflammation of the joint is accompanied by suppuration; the pus is produced either by the granulations, or else forms on the surface of the synovial sac which is not much diseased; sometimes in the same sac there is a subacute synovitis, while another part of the sac remains intact, and still another is completely degenerated; the knee and elbow joints are especially liable to these circumscribed separate diseases of individual synovial sacs, which only communicate with the cavity of the joint by small openings. These suppurations are usually accompanied by acute exacerbations of pain and fever, especially when the abscess opens externally, and synovial sacs, which have previously participated little in the inflammation, suddenly become acutely or subacutely diseased. An early profuse suppuration of a joint is sometimes an evidence of the previously *slight* degeneration of the synovial membrane, as most pus is given out by serous membranes in the stage of purulent catarrh. The pus from the synovial granulations is usually of slight amount, and of serous or mucous consistence. The symptoms may be different, if, as often happens, there be also suppuration in the cellular tissue around the joint, and *periarticular abscesses* (which, indeed, may occur without disease of the joints) accompany the fungous inflammation of the joints. All of these suppurations are important, from the fact that they impair the general health, partly by the loss of juices, partly by the fever.

Lastly, we must give some attention to the vital condition of the inflammatory neoplasia. The vitality, the luxuriance of growth, and the future fate of the chronic inflammatory new formations, greatly depend, as you already know, on the general constitutional condition of the patient; in fact, this is so to such an extent that from the vital condition of the local affection we may often make a decision as to the general health of the patient. Fungous inflammation of the joint with caries sicca, and a disposition to cicatricial contraction of the new formation, usually occurs in persons otherwise healthy, and in these cases it is often difficult to find any cause for the chronicity of the disease, which was said to have been first induced by cold, fatigue, or injury of some sort. We also find the most luxuriant, spongy granulations and secretion of muco-pus in tolerably healthy, or at least well-nourished persons, in fat, scrofulous children, also as the chronic continuation of an acute articular inflammation in persons previously healthy, who have become anæmic from the long suppuration. Great tendency of the neoplasia to break down into pus, or to molecular disintegration, is usually a sign of bad nutrition; we find thin, badly-smelling pus in large amounts, with excessive ulceration of the skin, and fistulous openings, that look as if cut out with a punch, in the articular inflammation, with or without caries, of old cachectic patients, in badly-nourished tuberculous subjects and scrofulous children. Here we may have the same course of affairs as in torpid caries; the neoplasm is very short lived, it breaks down almost as soon as formed; and along with the caries we have necrosis, as in the small bones of the wrist, more rarely in the epiphyses, also caseous degeneration of the neoplasm.

FIG. 96.



Atonic ulceration of the cartilage from the knee-joint of a child; the cartilage-cells, which only proliferate slightly, undergo fatty degeneration, and they, with the intercellular substance, break down very rapidly. Magnified 250 diameters.

We could distinctly separate this *atonic* form of chronic suppurative inflammation of the joint from the fungous variety, but avoid doing so: first, that we may not disturb the general description; secondly, because this form also often begins as a typical fungous synovitis, and subsequently passes into the torpid form as the nutritive state of the patient declines. We find it chiefly on autopsy, and should altogether mistake the earlier stages if we did not study the disease in resected and amputated joints. I shall not continue the anatomical details, which might be carried much further, but what has already been said will suffice to explain to you any given case. It is not impossible to group the different modifications of the above processes in classes and to analyze them separately; but this seems to me of no practical value, for these forms offer at present no special etiological, prognostic, or therapeutic features. If you correctly understand the anatomical course and recall my description in all cases that you see living or dead, in resected or amputated limbs, you will soon understand the disease and require no further classification of its symptoms.

About the *causes* of chronic fungous articular inflammation there is little to say beyond what you already know. The scrofulous diathesis especially predisposes to it; acute, spontaneous, or traumatic (whether from wounds, contusions, or sprains) inflammations of the joint occasionally become chronic. Scrofulous children, three years old and upward, are especially inclined to these joint-diseases; a fall or twisting of the joint often proves an exciting cause. Cases occur where we can find no local or general cause for the disease. In Switzerland I have very often seen atonic forms of fungous purulent inflammations of the joint in old people, where no cause for them could be discovered.

The course of this disease is very varied, but it is always chronic, lasting for months, usually for years; often interrupted by pauses and improvement, then again exacerbating. The disease may halt, and recover at any stage; in the first stages this recovery may be perfect, that is, the joint may remain entirely movable; or it may be imperfect, that is, more or less stiffness of the joint is left. Before the cartilage has commenced to proliferate, or has its under surface disturbed by any neoplastic tissue growing from the bone, there is a possibility of tolerably good motion being restored—which, however, may be impaired by cicatricial contraction of the fungous synovial membrane, and of the infiltrated ligaments, as well as by secondary contractions of the muscles. If the cartilage be partly or entirely destroyed, and caries has occurred gradually or with the onset of the disease, it may recover with anchylosis, the cartilage is not restored; the granulations of the adjacent surfaces of cartilage gradually unite, and often firm adhesions form, which may even ossify. Whether the disease goes on so far or the destruction of the joint continues to progress, depends greatly on the constitution of the patient; treatment may be of great benefit, if begun early. The extent to which the muscles sympathize varies greatly; according to my experience, the highest grade of muscular atrophy occurs in those cases where there is no suppuration of the joints but caries sicca, and where the joint-disease seems to proceed from ostitis.

Now for a short discussion of certain symptoms. Each form of this disease may run its course with more or less pain; the cause of this I am unable to explain; there are cases where the bone is extensively destroyed, without any pain, others where it is very severe; the acute exacerbations with development of new abscesses are always rather painful—on probing the fistulæ we sometimes find bone, at other times not; whether we feel it or not, depends on whether it is covered with granulations or lies exposed; the same is true of friction; crepitation is only valuable as a sign of caries of the articular extremities, when it exists; if it fail in the later stages, it is no proof that the bone is not diseased. The deformity, the displacement of the articular surfaces, pathological or spontaneous luxations, are the only evidence at all certain of the extent of the destruction of the bone; here we can only be deceived when the capsule has ruptured early, and the head of the bone is actually luxated; a rare case, which has, however, been seen in the hip, and might possibly occur in the shoulder. In regard to judging of the anatomical condition of the joint, little can be added to what has already been said, but we have some assistance from the etiology and duration of the complaint. Profuse suppuration from the joint is always a sign that part of the synovial membrane has not yet been destroyed, or that there are large abscesses near the joint; the secretion from fungous granulations is less abundant, serous or mucous. We have no certain evidences of the extent to which the cartilage is destroyed. To add any thing about the *diagnosis* and *prognosis* would only be to repeat what has already been said, from which you have all the data for forming your judgment. From my own experience, I think I may say that slight swelling of the joint, with great pain and early muscular atrophy in anæmic children, but with little or no suppuration, indicates primary disease of the bone, and renders the prognosis very bad. A good nutritive condition is the most important point for a favorable prognosis, which would not be very greatly affected even by early and extensive suppuration.

## LECTURE XXXVIII.

Treatment of Tumor Albus.—Operations.—Resection of the Joints.—Criticisms on the Operations on the Different Joints.

Now let us take up the subject of treatment. As in all chronic inflammations, this must be both general and local, and the general treatment should be the more prominent, the more chronic and insidious the disease; it is unnecessary for us to waste words over this constitutional treatment, which will depend on the peculiarities of each case; you already know its outlines. The salient points for treatment are, the nutritive state of the patient, the quality of his blood, and the general hygienic and dietetic conditions under which he lives. It is your duty conscientiously to advise your patient to the best of your knowledge, but you will soon find that on these points you meet the greatest indifference, and that your advice will rarely be followed. The worst instances, such as hereditary predisposition, we shall not be able to affect; for we can never expect to choose the strongest persons out of healthy families for the propagation of the species, and to forbid marriage to feeble persons from sickly families. Regarding the local treatment and its results, we may say, in general terms, that it is the more effective the more acute the stage; as a rule, it is not difficult to relieve subacute exacerbations. or subacute commencements of the disease. In these cases we derive great benefit from the already oft-mentioned remedies: strong salve of nitrate of silver (ʒ j to ℥ j of lard), painting with tincture of iodine, flying blisters, wet compresses, gentle compression with adhesive plaster; this should be accompanied by absolute rest of the joint, which in the lower extremities can only be attained by continued confinement to bed.

As yet I have no experience of the value of massage in the commencement of tumor albus; it should be used with great care. I cannot help thinking that massage used too strongly in these cases might induce suppuration where there was any tendency to it; so I would only recommend this treatment in torpid cases.

[In all cases if we do not apply massage to the diseased joint we may to the muscles of the limb to prevent their atrophying, which would otherwise result from continued rest.]

There is no doubt that in most cases of commencing and progressing diseases of the joints traction is more efficacious than plaster bandages ; hence, in my clinic you will see it used more frequently ; but in private practice you can not persuade all patients to go to bed, and, moreover, the method requires such careful watching from the surgeon as to interfere with its employment. *Taylor*, an ingenious American surgeon, has constructed splints for the lower extremity by which traction may be so applied as to free the joint from pressure and enable the patient to go about. These splints often act excellently, but they are difficult to make, and their use requires a certain experience on the part of the surgeon. All of these mechanical aids—plaster dressings, supports, traction, *Taylor's* splint, etc.—require continued supervision to prevent injury from pressure and friction and from displacement of the apparatus. In the case of children great patience and perseverance are needed in judging whether the extension is enough or too great, to accustom them to the inconvenience of the apparatus, to quiet the anxiety of the parents when the child cries, and by friendly talk or sober, earnest efforts to make the children obedient and prevent their loosening the apparatus. This treatment can rarely be thoroughly carried out in private practice ; hence, treatment in hospitals or orthopedic institutions cannot be too strongly urged, at least until the chief dangers from deformity are passed.

With such a dressing we may permit the patient to go about, if it does not pain him ; in so doing, he may use a cane or crutches, according to the weakness of the affected limb. Should the patient need baths at the same time, the bandage may be divided longitudinally, and be removed before the bath and replaced subsequently. This treatment has the advantage that the patient uses the muscles of the extremity somewhat at least, and consequently they do not entirely atrophy ; we are not to think that stiffness of the joint must necessarily result from wearing the plaster-splint for a length of time ; we not unfrequently find the opposite, that is, that a limb which was very slightly movable before the application of the dressing is more so afterward ; this is because the swelling of the synovial membrane often subsides under the bandage. Before applying the plaster-dressing we may rub the limb with mercurial ointment, or apply mercurial plaster, or even rub in the nitrate-of-silver ointment. In all chronic cases of fungous inflammation of the joint, I cannot sufficiently recommend to you the plaster-splint; this treatment appears very inefficient, yet it is more useful than all the other remedies that we have for combating this disease. I can assure you that, since following this treatment perseveringly, my cases are less frequently complicated with suppuration and fistulæ. Even when there is evident fluctuation you may apply the dressing; it is true you will rarely see the abscess reabsorbed, still, when it opens spontaneously under the bandage, as the patient will readily notice from the moistening of the dressing, this will take place more quietly, propitiously, and painlessly, than under any other plan of treatment. When fistulæ have formed, we may still use the plaster-splint, simply slitting it up and putting in new wadding; it should be removed daily and the sores dressed, then reapplied; at the same time the constitutional treatment should be persevered in. If the limb be very painful, and there are any fistulæ present, we should use splints with openings. In this way I have occasionally preserved a good, useful position in joints moderately movable, where the prognosis was at first very bad, and have indeed been frequently most agreeably surprised at the results of this treatment. Extension must be undertaken very carefully in joints that are suppurating or much diseased in any way, and, if even during anæsthesia there should be resistance, complete extension should never be made at one sitting, but it should only be carried so far as may be done without great force. In knee and hip diseases I use, with great benefit, the extension by weights which has been so often recommended, and occasionally thus prepare patients, especially children, for the application of the plaster-bandage. *Volkmann* deserves many thanks for his energetic recommendation of this plan of treatment, which he calls the "Distractionsmethode." He attaches great importance to the fact that the extension reduces to a minimum the pressure of the articular surfaces on each other, that is caused by the tension of the muscles and contraction of the ligaments. The mode of applying the extension is so very important for the practical use of this method, that I must particularly recommend you to give you special attention to its mechanical application in the clinic.

Perseverance on your part and on that of the patient is absolutely necessary, for the cure of chronic inflammations of the joints; represent to the patient, at the outset, that this is a disease of at least several months', possibly of some years' duration, and that the dressing is not to be left off till the limb is free from pain, and strong enough to walk on, whether motion be lost or not. Regarding cold abscesses, I repeat the advice only to open them, when you propose to follow them at some time by an operation; if this cannot be done, or you do not intend to do it, leave the opening to Nature, even if it should require years.

So far, I have briefly given you my maxims regarding the treatment of fungous inflammation of the joint, but I must not neglect to call your attention to the fact that other surgeons have different views on the subject. There are still advocates of the strong classical antiphlogistic treatment, who, even in chronic inflammations of the joints, from time to time apply leeches or wet cups, put on compresses with lead-water, and give cathartics; later they use cataplasms, and finally moxæ and the hot iron. If the disease continues to advance, if fistulæ have formed here and there, if the patient has become very anæmic, they consider amputation indicated, especially when there is crepitation in the joint. This was the old belief; the results were generally unfavorable or favorable, as we may choose to consider them; that is, they were the latter so far as regards the favorable course of the amputation, which was made, sooner or later, under such circumstances. Even now it astonishes me to see how often amputations of the thigh are made for tumor albus of the knee, in many hospitals; it is not saying much to mention that, in my own hospital service, I have rarely found thigh-amputations indicated for caries of the knee; but it appeared to me very remarkable that, during the seven years I was assistant in the surgical clinic at the University of Berlin, there were only two amputations of the thigh for caries of the knee, while formerly, in the reports of the smallest hospitals, several such amputations were reported every year. I am much inclined to refer the more favorable results, the rarer indications for amputation, to the treatment of the disease by the plaster-bandage, which was chiefly introduced and persistently carried out by *Von Langenbeck;* and I am firmly convinced that, by it, a large number of limbs have been preserved in a relatively good condition, which, in former times, would certainly have been amputated. I would not recommend the abstraction of blood in chronic disease of the joints; it can only prove beneficial in subacute exacerbations, and in these very cases we have better remedies, which are not at the same time injurious; for it is certainly improper to abstract blood once, or even oftener, from patients who are inclined to anæmia by their disease itself. In some cases of subacute attacks in chronic inflammation of the joints, cold is an excellent application; in such cases I now use ice with good results; but I cannot say that cold would be particularly beneficial in cases that run their course without outward symptoms of inflammation; and it is no slight affair to treat a patient with ice for years, keeping him in the same position in bed with a bladder of ice on his knee, which, at any rate, does not give him much pain. *Esmarch* claims very favorable results for persevering treatment with ice. Now I must speak of the *persistent application of heat*, which may be accomplished by the careful application of cataplasms, compresses wet with warm water, or even the continued use of warm baths for weeks. This treatment may be indicated when the course of the disease is exceedingly torpid, when bad-looking fistulous ulcers, deficient vascularity of the granulations, or bad, thin secretion, seems to indicate a moderate irritation of some kind. However, when high temperatures are applied, they should not act too long, or their effect will be lost, and there will be complete relaxation of the parts, instead of the fluxion that it was proposed to excite.

From the above description of the benefits of treatment, you may see that in fungous inflammations of the joints the results are generally good, if we leave out of consideration the greater or less stiffness of the joint which remains; this is particularly the case if the patient is treated early. Still, some cases are not cured, in spite of the most careful treatment; this is partly due to the anatomical condition of the joint, partly to the general health of the patient. For anatomical reasons, disease of the joints of the hands or feet is the most unfavorable; from the many small bones and joints affected, the progress is usually excessively tedious; the disease may begin quite chronic at one of the small joints of the hand or foot, may remain stationary at this point for a time, then spread to the next two, again halt a while, or even recede; but a new joint is attacked; suppuration begins first in one place, then in another, the patient grows anæmic and weak, he is condemned to inaction for years, and finally longs to have the affected limb amputated, so that he may once again feel well, after his years of suffering. In other cases a scrofulous or tuberculous cachexia gradually induces anæmia, indigestion, fatty degeneration of the internal organs, tuberculosis of the lungs, etc., so that from the general health of the patient we must give up all hopes of a cure. If, under such circumstances, we leave the disease to itself, the patients die after years of suffering; the end comes the sooner the larger the joint affected (knee, hip), and the greater the number simultaneously affected, as is apt to be the case in scrofula and tuberculosis. Under such circumstances we may resort to two modes of treatment: 1. Give up the limb to save the life, that is, *amputate;* 2. Give up the attempt to cure the joint-affection, cut out the diseased ends of bone, so as to save both life and limb, that is, *resect* the joint.

Comparing these two remedies theoretically, there can be no doubt that resection is preferable to amputation, and in principle this is certainly true; modern surgery is justly proud of the institution of resection of joints. Nevertheless, certain circumstances may combine to render amputation preferable in any given case; chief among these is the state of the patient's general health. After resection of the joint we have left a large wound with two sawed edges of bone, which will certainly continue to suppurate for weeks, possibly for months; there may be suppuration of the subcutaneous tissue, of the sheaths of the tendons, and suppurative periostitis and necrosis of the sawed edges, things which patients may live through, but which always require time and strength. If, then, in badly-nourished, cachectic persons, loss of strength should indicate operative interference, amputation is often a more certain remedy for saving life than resection. The surgeon should always think more of saving the life than the limb. We have also to answer the question, Can the patient bear resection, with its sequelæ? It is difficult to give a general answer to this question; even in individual cases a decision may be difficult: we must determine whether the patient is emaciated, anæmic, and debilitated, simply by the drain on his system, or if there be more serious lesions of internal organs; in the latter case amputation would be preferable, if, indeed, any operation would be serviceable. Of course we do not operate on atrophic children with disease of several joints, cold abscesses, diarrhœa, aphthæ, etc., or on persons with tuberculous cavities in the lungs, or with indurated, fatty liver and spleen, or on old marasmic individuals; we cannot give any aid to such patients. But a still more important question is, Which operation is less dangerous to life? We cannot give a *general* answer to this question; we must separately consider the joints concerning which the question of resection arises. In caries of the *shoulder*-joint resection is less dangerous than disarticulation of the arm at the shoulder-joint; the same is true of the *hip-joint;* hip-joint amputations are among the most dangerous in surgery, while in young subjects resection is not so very fatal. Hence we are not to think of exarticulation at the shoulder or hip on account of caries; here the only question is, Is the general health of the patient such that we should let the disease run its course, or shall we arrest it by resection? In the most favorable cases of spontaneous cure there will be anchylosis in a bad position; if recovery takes place after resection, the extremity remains movable at the shoulder or hip joint. These chances speak strongly for resection, especially at the shoulder-joint; here we might decide on resection quite early, even in order to get the patient about soon and in good order. Resection of the hip is open to one grave objection: we cannot resect the acetabulum, which is usually diseased at the same time, or we can only do so imperfectly; hence, when the joint is much diseased, the resection is imperfect; slighter grades of the affection may even recover without operation.

In the *elbow-joint* the state of affairs is more favorable, perhaps the most favorable; the resection of this joint is not more dangerous than amputation of the arm; but, in favorable cases, after resection, quite a useful joint is left, while after spontaneous recovery there is generally anchylosis; in these cases the choice is easier: we prefer resection of the elbow-joint, not because the operation must be done to save life, for caries of this joint is only dangerous from long duration, but because, while the danger is relatively slight, it offers good chances of motion, and in any other case there is usually anchylosis; indeed, the anchylosed joint has even been sawed out in order to obtain a movable false joint. Unfortunately, more recent observations on the motility of arms with resected joints have shown that the false joints formed after operation become more relaxed in the course of years, so that finally the operated extremity does not remain as useful as was formerly supposed. The case is very different with the *knee-joint;* here resection is quite a dangerous operation, being on a par with high amputations of the thigh; after resection of the knee we only obtain anchylosis, which is also the result of spontaneous recovery. Now, as this operation is quite dangerous, and as it gives no better results than non-operative treatment, in case the disease is arrested, it should only be done to save life, and, even in this respect, it is of doubtful advantage. I have rarely decided on an operation for caries of the knee-joint, either for amputation or resection; we can only propose amputation when all treatment is fruitless, and the patient is failing rapidly, or when it is an old person in whom extensive caries of the joint would be very unlikely to heal.

The above are my personal opinions, which constantly become more fixed, as I see more such knee-diseases recover spontaneously. I have seen many children die of coxitis, and consequently am rather in favor of resection of the hip, in spite of the want of success of my own operations; the only deaths I have seen from caries of the knee have been in old, marasmic persons and those with tubercles and extensive cavities in the lungs, while they have been rare in children; in all of these cases operation would have been useless. Here you have my belief about operations of caries of the knee. Other surgeons have different opinions; in England, especially, the operation is so popular that it is very often performed. I believe that many German surgeons share my views on this subject, others are more undecided, as they view this operation more favorably from having seen a few successful resections of the knee-joint. Formerly, I was entirely opposed to resection of the knee-joint, but have been rather unsettled by a series of favorable results that I have lately had from this operation. If the cases with good chances be chosen for operation, and unfavorable or doubtful ones never operated on, the operations will be mostly successful, but few patients will be cured. The same is true of most great operations; if one has some experience, and does not hesitate to send most cases away uncured, interesting himself only in the favorable cases, he may soon attain the reputation of a very fortunate operator. Many eminent surgeons deceive themselves in this way.

Now we come to the *wrist-joint;* here resection usually consists in the removal of all the bones, and sawing off the lower surfaces of the radius, perhaps also those of the ossa metacarpi. I have performed this operation several times, occasionally with brilliant results, the hand becoming perfectly movable and the fingers useful; two of the patients were seamstresses, and were able to resume their occupation, the third and fourth unfortunately lost patience; after the operation, when the wound had closed except two fistulæ, and the pain had ceased, they stopped treatment; there were still some carious spots in the metacarpal bones which should have been extirpated, when the result would certainly have been as good as it was in the previous cases. I should have liked to resect the hand more frequently, but several times have submitted to the patient's special request to amputate the forearm. It must seem strange that a patient does not readily consent, when the surgeon proposes, by a tolerably safe operation, such as resection of the wrist, to preserve the hand; I always felt obliged to say that it would be several months before the wound healed, so that the patients should not expect too much; they replied that it was too long a time, they had not used the hand for four, five, and eight years, and it always pained them; they were tired of treatment, and had decided to lose the hand, so they would not again undertake a long course of treatment. I have told you this that you may see what obstacles the surgeon runs against when he honestly tries to do the best. All the cases of caries of the wrist are by no means suited for resection; we never decide on an operation before there is extensive destruction of the bones, although we know that caries of the wrist very rarely spontaneously recovers with movable joint. Caries of the wrist is not frequent as compared with that of the knee and hip, and is particularly rare in children, being more frequent in adults. The cause of the difficulty of recovery is partly due to local conditions which we have previously described. Besides this, there are about the hand so many tendons, most of whose sheaths participate in the disease; the fingers are stiffly extended, the metacarpal bones, radius, and ulna, are also frequently diseased, though they may be only affected with periostitis. The other soft parts about the hand, especially the skin, are perforated by numerous fistulæ, or even extensively destroyed, so that the most favorable circumstances for resection do not exist. Hence, where extensive caries of the hand is accompanied by considerable degeneration of the neighboring parts, amputation of the forearm will justly assume its old position. Extraction of single metacarpal bones, or simply sawing off the radius, is rarely sufficient; I have, indeed, seen cases where the disease was limited to one or two metacarpal bones; these had become necrosed, and the disease terminated at that point; one patient was sent to me for amputation of the hand, and was much pleased when, after examination, I told him that amputation was not necessary. But these cases are rare; usually the disease advances, and is not arrested by the extirpation of the bones which are chiefly diseased. I think that, on the whole, total resection of the wrist is still too little employed; according to my experience, it is worthy of the greatest attention from surgeons. This operation, as well as a similar one on the foot, of which we shall speak shortly, is well supported by a reasoning that has been falsely applied to resections in general; i. e., if resection does not arrest the local disease, we may still amputate. In resections of the hand and foot this is true, and they are rarely followed by pyæmia, but the case is not the same with the shoulder, hip, elbow, and knee. If these operations are unsuccessful, if suppuration be exhausting, or pyæmia occur, we can hope little from amputation or exarticulation. Lastly, we come to the ankle-joint, comprising the joints of the tarsus as well as the tibio-tarsal articulation. The circumstances here very closely resemble those for the wrist; although caries of single bones, as the not unfrequent caries necrotica of the calcaneus, will spontaneously recover with time, especially in children, just as scrofulous caries of the fingers, toes, metatarsal and metacarpal bones do, even in young adults, caries of the joints of the foot rarely recover spontaneously, and in old persons hardly ever do so. Consequently, in these cases operation will frequently be indicated at some stage of the disease, and on superficial observation we might think that resection and extirpation of bone should be very commonly resorted to; but, practically, there are two objections to the extensive resort to these operations in caries of the foot: 1. The experience that, after extirpation of one bone, the disease often attacks another, and consequently perfect recovery does not result. 2. The fact that the foot must always retain sufficient firmness for the patient to walk; so, while we may remove the cuneiform bones, the scaphoid and cuboid, or even the astragalus or calcaneus, if we remove *both* the latter bones, and perhaps also saw off the articulating surfaces of the tibia, we should have a rather useless foot, which would be worse than a good stump. The cicatrices occurring at the place whence the bone was extirpated contract greatly after a time, and even if some bone form in this cicatrix, still it is not regenerated as after necrosis, but the foot contracts greatly at the point from which the bone is absent, and thus becomes distorted and useless. These are decided objections; moreover, a good stump, such as is left by *Chopart's* or *Pirogoff's* operation, is often just as good or even better for walking than a weak, deformed foot, and it requires several months to get the latter into shape, while the former may be obtained in six to eight weeks. In one case, I removed all three cuneiform bones, and the os cuboid, with good results; in other cases, in boys, I have removed the astragalus; then the tibia articulated with the calcaneus, the new joint remained movable, and the patient did not even limp; such results are very encouraging for this operation. Another time I wished to remove the calcaneous alone for caries, but unexpectedly found the lower part of the astragalus affected, and had to remove it also; the result was miserable: the young boy lay six months in the ward, and even then did not recover, so I amputated at the lower part of the leg, and the wound healed by first intention; a few weeks later, the patient left the hospital well, with a good wooden leg, glad to be rid of his sore foot. The very favorable results of *Pirogoff's* amputation make a strong opposition to resection of the ankle-joint, and I think that experience will soon speak more strongly than now against too great employment of exsection, and for amputations through the foot.

Resections of joints, which have excited so much controversy the last twenty years, at first appeared so brilliant from the favorable results in certain joints, such as the elbow and shoulder, that they were sometimes too much resorted to; this is the fate of all inventions of the human mind. We are only now gradually coming to certain indications for these operations; of course statistics had first to be collected, and it was soon found that resection was of varied value in different joints. Although I am not prepared to say that the question is even now settled, still I believe I have given you a correct *résumé* of the present position of affairs.

I cannot refrain from making one observation at the close of this chapter. In the Canton Zürich patients who had been successfully treated for caries, by resection or amputation, often returned, and, sad to say, many of them who, after suffering for years, had been perfectly cured, and had left the hospital quite strong, came back after a year or two with caries of other bones, or with tubercles of the lungs, and often died there. I have been unable to gather any extensive statistics as to the final terminations of bone and joint diseases, but fear that they will prove much more unfavorable than we generally incline to believe.

[Instead of simple compression, you may render the extremity entirely bloodless by means of *Esmarch's* bandage; at first this anæmia may be kept up five minutes, and subsequently for a quarter or half an hour. Regularly repeated daily, in the early stages of tumor albus, this treatment has given me very good results.

When cold abscesses have developed, under the present perfected system of antisepsis, you may open them, if you are sure of being able to reach the whole extent of the abscess-cavity. You divide the soft parts, let out the pus, and, with the sharp scoop, scrape the inner surface of the cavity, fill it with iodoform, unite the wound, except at the points for drainage, and apply *Lister's* dressing. The limb should be kept immovable. Should you await the spontaneous opening of the abscess, it probably will do no harm.

It remains to be seen whether the statistics of knee-joint resections will be decidedly changed by the employment of iodoform; experience thus far is encouraging.

Let me call your attention to the etiological relation of chronic bone and joint affections, and acute infectious diseases of children, such as the acute exanthemata, diphtheria, pertussis, etc.]

Less attempt is made than formerly to obtain movable false joints after resection; but more frequently we seek by partial removal of the bones chiefly diseased, under *Lister's* method, and with the least possible suppuration, to cause anchylosis of the joint.

Unfortunately, return of the disease is not rare, even in joints which had been healed by anchylosis for years. Persons who have suffered from the above forms of chronic articular inflammation rarely attain old age. You will find few persons above forty or fifty years old with anchylosis from tumor albus. This seems another proof that these diseases are associated with some constitutional taint, difficult as it is in all cases to prove this, and to demonstrate it to those who are inclined to explain all diatheses and dyscrasiæ as vague theories of old physicians.

## LECTURE XXXIX.

*B.*—Chronic Serous Synovitis.—Hydrops Articulorum Chronicus: Anatomy, Sy toms, Treatment.—Typical Recurrent Dropsy of the Knee-Joint.—Appen Chronic Dropsies of the Sheaths of the Tendons, Synovial Hernias of the Jo and Subcutaneous Mucous Bursæ.

### *B.*—CHRONIC SEROUS SYNOVITIS.—HYDROPS ARTICULORUM CHRONICUS.—HYDRARTHRUS.

The chronic diseases of the joints that we have now to describe are much more rare than fungous synovitis and its results, which we have already described; taken altogether, they are scarcely so frequent as the former, and, as a body, they form a decided contrast to suppurating inflammations of the joints, for they never spontaneously suppurate, they only do so when acted on by repeated irritations, injuries, etc. We shall commence with the most simple of these forms, with chronic serous synovitis, or hydrops articulorum chronicus, or hydrarthrus. The disease consists in a morbid, slowly-increasing collection of rather thin synovia; the synovial membrane changes very little, it gradually becomes somewhat thicker and firmer, the connective tissue increases, but without any marked increase of vascularity; the tufts elongate, and, although the vessels form into loops at their apices, the substance retains the firmness of connective tissue, while from plastic and serous infiltration it grows soft and resembles granulations in fungous synovitis. In serous synovitis this does not occur; the entire pathological changes of tissue are very slight, even when the disease has lasted a long while. Some surgeons wish to consider these dropsies of the joints, as well as similar diseases of the mucous bursæ, as not belonging to the chronic inflammations, but as constituting peculiar diseases. This does not seem to me justifiable. No one will dispute that chronic catarrhs of the mucous membranes, with a tendency to hypersecretion, are to be classed among the chronic inflammations; chronic dropsy of the synovial membrane is perfectly analogous to chronic catarrh of the mucous membranes.

Chronic dropsy of the joints is often the remains of an acute articular dropsy, caused by contusions, catching cold, etc., as has already been described; but in many cases, also, the disease is chronic from the start, and remains so. Hydrarthrus is most common in young men, and occurs most frequently in the knee-joint; it often comes on both sides; it is very rare in the shoulder, hip, or elbow; I have never seen a pure case of it in the other joints. When the disease is well advanced it is readily recognized, and even the laity know it as "dropsy of the joint." The joint is much swollen, fluctuates all over; in the knee we have also the motion of the patella; it is lifted up by the fluid, and may be readily pressed again into the intercondyloid fossa, occasionally with a perceptible sound. As the surfaces of the joint are united by firm ligaments (in the knee by the lateral and crucial ligaments), which are not so easily stretched, the fluid collects chiefly in the mucous bursæ adjacent to the joint, and on this account we may often diagnose the swelling as synovitis by simple inspection, especially in the knee-joint, where the bursæ under the tendons of the extensors at both sides of the patella, and in the popliteal space, are greatly distended by the fluid; while, on the other hand, in regular swelling of the capsule, the enlargement is regularly round. Sometimes, also, patients with this disease can move their joints quite freely and without pain; they can often walk quite a distance, and occasionally have so little inconvenience that they do not ask advice of the physician; even examination of the joint by palpation is painless. Where the dropsy of the joint is considerable, great exertion readily causes fatigue of the limb, as well as pain and increased exudation; however, after resting a while, this passes off, and generally the inconvenience is very slight.

The prognosis is good in so far as these dropsies of the joint lead to nothing further; the fluid may increase enormously, but that is all; unless there be some overstraining or injury, the disease remains the same. As regards recovery, the prognosis is most favorable in those cases where the disease remains after an acute or subacute commencement; in these cases, as a rule, complete recovery takes place by reabsorption, although it may be slow. On the other hand, those cases where the disease is chronic in its commencement and course are very obstinate, and are often extremely difficult to cure.

The treatment consists in the application of the remedies already described, which are to be perseveringly used while the joint is kept at perfect rest, viz., tincture of iodine, flying blisters, and compression. The latter is the most effective remedy, but it must be strong and continued (forced compression, according to *Volkmann*); we may apply firm dressings with moist or elastic bandages; the patient must lie still during the treatment; if there should be any œdema of the leg, it will do no harm, but, if the toes grow blue and cold, the bandage must be removed. If the patients will not submit to this treatment, we may let them wear a large mercurial plaster, with a snugly-fitting knee-cap of leather with elastic insertions, which prevents too much motion of the joint, and gives the limb more firmness and security in walking. If all this treatment does no good after months or years, or if the improvement has only been temporary, we may still resort to simple tapping, or to tapping, followed by injection of iodine.

Usually simple tapping does little good. You pass a fine trocar into the joint alongside of the patella, allow the fluid to flow out slowly, and close the canula a little before it has all escaped, so that no air may enter the joint, then cover the wound with adhesive plaster; now paint the joint with tincture of iodine and envelop it with wet bandages or a collodial bandage, and in some cases you may attain a cure; there will be a rapid collection of serum and some pain in the joint; this new fluid may be completely absorbed. If this operation has done no good, if the fluid collects again to the same amount, and remains unchanged, you may make the tapping followed by injection of iodine. This operation is not free from danger; you perform it as follows: First tap the joint carefully, as above directed, then fill a well-made syringe with a mixture of officinal tincture of iodine and distilled water in equal parts, or, if you wish to be very careful, one part of tincture of iodine to two of water; after seeing that there is no air left in the syringe, you may inject from one to two ounces of this mixture, according to the amount of previous distention of the joint; keep the fluid in the joint three to five minutes, according to the pain induced, then let it escape slowly; now carefully close the wound, and make compression, as above described. A new acute serous exudation always results; this remains stationary about eight days, and is then slowly absorbed, and recovery usually follows. Of course, under such treatment, as after simple tapping, the patient must remain absolutely quiet, for there is always inflammation, and perfect rest is the first requirement in inflamed joints. It is not quite evident why it happens that, when tincture of iodine comes in contact with a serous membrane which was disposed to excessive secretion, even for a short time, it should have such an influence in altering and arresting the secretion; formerly it was thought that after these injections, which were advantageously used in many chronic dropsies of serous membranes, there was adhesive inflammation, a union of the surfaces of the serous sac, and its consequent obliteration; this is by no means the case, at least after the successful injections of iodine in hydrops articuli; if such adhesions occurred here, the joint would become stiff. What really occurs is as follows: The iodine is deposited in the surface of the membrane and in the endothelium; it remains here for months, at least, and by its presence appears to prevent further secretion. At first there is strong fluxion with serous exudation (acute serous synovitis), but the serum is again absorbed by the still-distended vessels, and subsequently the membrane shrinks to the normal volume by condensation of the connective tissue, which subsequently remains more dense. So we may consider the process of cure as analogous to the similar process in the tunica vaginalis propria testis, in the cure of hydrocele of the tunica vaginalis, or water-rupture; after injections of iodine in hydrocele, there has been an opportunity of making many examinations, from which the course of the cure appears to be as above stated; the shrinkage of the serous membrane, with new formation of endothelium, seems to me to be the final cause of the arrest of the secretion.

Iodine injections in hydrarthrus are made by few surgeons; I have seen them made three times, and have made two, always with good result; but this is not always the case; then they must be repeated, but I warn you against repeating them too soon: you should at all events first allow the acute stage after the operation to subside. Cases have also occurred where severe inflammations of the joint have resulted after these iodine injections, which have been most used in France because they are a French invention (of *Boinet* and *Velpeau*); as so often happens in traumatic articular inflammations, the acute serous synovitis became purulent; in favorable cases there was recovery with anchylosis, in some cases amputation was necessary, in other cases the patients died of pyæmia. These unfortunate terminations of an operation done for a disease, which is obstinate it is true, but not dangerous to life, have justly rendered injection of iodine into the joints unpopular; it is always dangerous to the joint and to life, and hence should be done as rarely as possible.

The *diagnosis* of hydrarthrus is usually simple, and the disease always very different from chronic fungous purulent synovitis; however, I would caution you that, in the commencement of tumor albus, also, there is occasionally a slight amount of serous exudation, and even fluctuation, in the joint, so that at first the diagnosis cannot always be exactly made; but observation for a few weeks suffices to show the nature of the disease, and, moreover, hydrops articulorum occurs chiefly in young adults, while tumor albus is most frequent in children.

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## APPENDIX.

### CHRONIC DROPSIES OF THE SHEATHS OF THE TENDONS, MUCOUS BURSÆ, AND SYNOVIAL HERNIAS.

We shall now say something of the chronic dropsies of the sheaths of the tendons. The disease consists in an abnormal increase of the synovia, secreted from the sheath of the tendon, for facilitating the motion of the tendon, and in abnormal distention of the sac. The sheaths of the tendons of the hand are most frequently affected. There is a gradual formation of a swelling in the hollow of the hand and lower end of the volar side of the forearm; and we may distinctly feel the passage of a fluid in the sheath of a tendon from the vola manus to the forearm, under the ligamentum carpi volare and back again. The fingers are generally flexed and cannot be fully extended; the movements of the hand and fingers are somewhat limited; there is not necessarily any pain, and the patients do not usually apply to a surgeon till the disease has attained a high grade.

Another form of this disease is partial hernial ectasia of the sheath of the tendon, with dropsy. On the sheath there forms a sac-like protrusion, about the size of a pigeon's egg, containing an abnormal amount of synovia of the sheath.

FIG. 97.

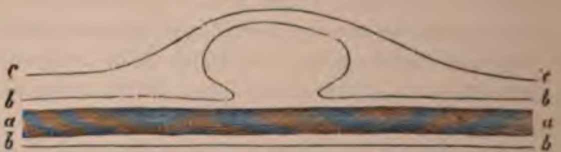


Diagram of the ordinary ganglion. *a*, tendon; *b*, sheath of the tendon with dropsical hernial protrusion upward; *c*, skin.

In ordinary surgical language this is called a *ganglion* when it comes on the back of the hand. It is of far more frequent occurrence than dropsy of the whole sheath of the tendon, but it only comes at certain places. Ganglia are most common on the dorsal surface of the wrist, where they arise from the sheaths of the extensor tendons; they are more rare on the volar surface of the hand and higher up the forearm, rarer still on the foot, where I have found them most frequently on the sheaths of the peroneal tendons. These ganglia usually contain a thick, mucous, vitreous-looking jelly. The contents of previously-described extensive exudations in the sheaths of the tendons may also consist of clear jelly; but frequently there are also innumerable white bodies, like melon-seeds, which are not organized, but usually consist of pure amorphous fibrine. These bodies may be present in such numbers that no fluid can be evacuated on puncturing the sacs. Sometimes we can diagnose these fibrine-kernels beforehand, from their giving rise to a strong friction-sound, such as occurs in subacute inflammation of the sheath of the tendons.

In the *treatment*, we must, above all, bear in mind that we should avoid any operation that might induce suppurative inflammation of the sheath of the tendon, and might disable for a long time or possibly cause a stiff hand in a patient who had been but little inconvenienced by his ganglion. Remedies, such as mercury and iodine, which so stimulate reabsorption in cases of acute or subacute inflammation, are of little use here. The simplest and the most frequent operation is *rupture of the ganglion*. If, as is customary, the ganglion be on the dorsal surface of the hand, we take the flexed hand of the patient before us, place the two thumbs close together on the ganglion, and make strong pressure; this sometimes ruptures the sac, the fluid is effused into the subcutaneous tissue, and then readily reabsorbed. When this method succeeds readily, there is not much objection to it, except that it does not always cause a radical cure. The small subcutaneous opening of the sac soon closes, the fluid collects again, and the disease continues as before. If we cannot rupture the sac with the thumbs, it has been recommended to do so with a quick blow by a broad hammer; although this succeeds now and then, I would not recommend it to you, for if unskilfully done it may cause a severe contusion, whose consequences we cannot always master. When the sac is too thick to rupture with the finger, I employ *subcutaneous discision;* I pass a narrow, short, curve-pointed knife (*Dieffenbach's* tenotome) horizontally into the sac, and with the point of the knife make numerous incisions on the inner wall of the sac, I then draw the knife slowly out, meantime pressing the fluid out of the sac. I then at once apply a compress, envelop the hand and forearm in a wet bandage, to prevent any extensive motion, and have the forearm carried in a sling four or five days. Then the bandage is removed, the small opening is healed, and the ganglion does not usually return, as it is apt to do after simple evacuation. The entire hernial sac has often been entirely removed, sometimes successfully without subsequent inflammation, but at other times with suppuration of the sheath or loss of motion of the finger, so that I do not recommend this proceeding to you. The difference in result after extirpation of these sacs may depend on whether there is a large or small communication with the sheath of the tendon, or whether there be none; that the latter state does occur I have satisfied myself by examination of the cadaver; but I cannot say whether in such cases the sac near the sheath of the tendon is newly formed, or whether the opening, by which most of these herniæ of the sheaths communicate with the latter, has been obliterated in the course of time.

The treatment of extensive dropsies of the sheaths of tendons in the palm of the hand and forearm is much more difficult, since, for various reasons, subcutaneous discision is not available here, and resorbents are of little use; the only thing left is to try other methods, which often at least induce some suppuration. Take into consideration then whether it be really necessary to do any thing severe. If the disturbance be not so decided as to greatly interfere with the patient's business, you had better leave things alone. But, if something must be done, your choice is almost limited to two methods, viz.: an extensive incision and punction, with subsequent injection of iodine

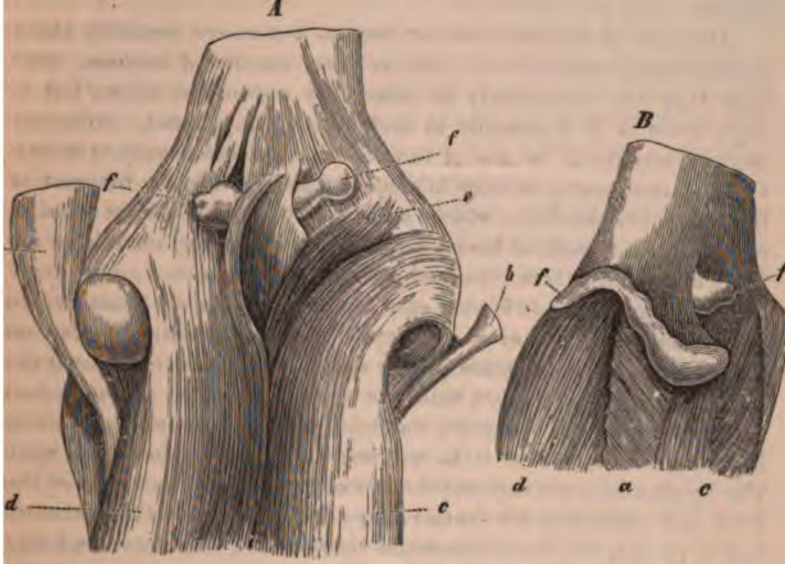
When you make the punction, which I prefer to incision, you should choose a trocar of medium size, as the fibrinous bodies will not escape through a very fine one. You will often have trouble in evacuating them even through a large canula; then you will facilitate the operation by injecting tepid water through the canula from time to time, so that the increased amount of fluid will aid the escape of the slippery fibrine-kernels. As already mentioned, the quantity evacuated is often large. I once took one and a half tumblerfuls from a tendon-sac. After all has been removed, fill a syringe with an ounce of a mixture of equal parts of water and tincture of iodine, or a corresponding quantity of solution of iodine and iodide of potassium, and inject it slowly. Let it remain in the sac one to two minutes, and then escape slowly. Now remove the canula, cover the wound with a small compress, bind up the hand and forearm carefully, and put it on a splint. The patient should stay in bed several days. The operation is followed by a considerable swelling, due to collection of fluid as a result of acute inflammation of the serous sac. If the tension become decided, we should remove the dressings, carefully close the puncture with plaster, then paint the swelling with strong tincture of iodine. In the more favorable cases, the swelling will then gradually subside, become less painful, and in the course of two to three weeks disappear entirely. In many other cases, however, there will be some, even if very temporary, suppuration, which may be checked and subdued with ice. In the worst cases there may be extensive suppuration of the sheath with necrosis of the tendon, and its results. Of course, opening the whole sac naturally induces suppuration.

On this occasion I must again repeat that there may be hernial protrusions from the capsule of the joint, just as from the sheaths of the tendons, which may become dropsical without the dropsy extending to the entire synovial membrane. The fibres of the capsule separate, and the synovial membrane passes between them into the subcutaneous tissue in form of the finger of a glove. Although such formations of round, pedunculated, long, wreath-like, and other shapes may develop from any joint, they are chiefly met in the knee, hand, and elbow; in the latter I have often seen these isolated dropsies of hernias of the synovial sac communicating with the joint; they are accompanied by slight stiffness of the joint.

I urgently warn you against operation on these ganglia of the joints; this operation may be followed by suppuration of the joint.

Cartilaginous bodies, enchondromata, sometimes even ossifying, occur in the tufts of the sheath of the tendons. Lipoma (*L. arbores*

FIG. 98.



Hernial protrusions of the synovial membrane of the knee-joint posteriorly (after *W. Gruber*). *A*. *a*, M. semimembranosus; *b*, M. biceps; *c d*, M. gastrocnemius; *e*, M. plantaris; *f f*, synovial hernias.—*B*. *a*, capsule of knee-joint; *c d*, M. gastrocnemius; *f f*, synovial hernia.

*cens* of *J. Muller*) has also been seen in the villi. The tumors should only be removed when they cause decided inconvenience.

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Here we shall also speak of fistulæ and chronic dropsies of the *subcutaneous mucous bursæ*. If one of these bursæ be opened by a simultaneous skin-wound, we often have protracted suppuration from the sac, which is not dangerous, it is true, although there may be an extension of the suppuration to the subcutaneous cellular tissue, which, from its duration, may prove very annoying; even after the greater part of the wound is healed, a fine opening remains; through this a probe may be passed into the sac; a moderate quantity of serum is daily evacuated through this fistula of the mucous bursæ. We may sometimes heal these fistulæ by cauterization with nitrate of silver and compression by adhesive plaster; but in some cases they are very obstinate. Then you may attempt, by injecting tincture of iodine, to excite a more intense suppuration of the inner well of the sac, and thus cause it to atrophy or become adherent; but a quicker way is to introduce a blunt-pointed knife through the fistula and slit up the sac and superjacent skin, so as to expose the whole interior; granulations will gradually spring up, and the wound will finally heal. I decidedly prefer this method.

*Dropsies of the subcutaneous mucous bursæ* are perfectly analogous to the above-described dropsies of the sheaths of tendons. Perhaps they may occasionally be caused by pressure or blows, but many cases it is impossible to find any exciting cause. Although dropsies may occur in any of the constant, or occasionally in newly formed subcutaneous mucous bursæ, they are particularly frequent in the bursa præpatellaris, which, according to *Linhart*, often consists of two or three mucous bursæ, lying over each other, sometimes entirely closed, at others communicating with each other. Dropsy of the bursa præpatellaris is very easy to recognize, for the tumor, which attains about the size of a small apple, is very evidently situated on the patella, and examination plainly shows that the sac containing the fluid does not communicate with the knee-joint. This disease often begins acutely or subacutely; the fluid collects rapidly, the swelling is painful, the skin over it is red, and the patient cannot walk well. The terminations are various; there is often entire reabsorption of the fluid, and a return to the normal state; in other cases the reabsorption is partial, the acute symptoms subside, and the state gradually becomes chronic. Rupture of the sac is one of the rarer terminations; this may be subcutaneous; the fluid is emptied into the subcutaneous cellular tissue, and induces diffuse inflammation. Rupture of both sac and skin is the rarest result; the disease then runs the course of a punctured or incised wound of the bursa, of which we have already spoken.

The form of the disease which is chronic from the start is more frequent than the acute. It begins slowly, without pain, and is more frequent in old than in young persons. In England this chronic dropsy of the bursa præpatellaris is called "housemaid's knee;" there it is said to occur particularly among the servant-women who have to scrub the stairs on their knees. But it seems to me very doubtful whether this has any effect on the occurrence of the disease, for it has been shown by many anatomists that in a kneeling position the weight of the body does not come on the patella, but on the condyles of the tibia. To bring the anterior surface of the patella on the ground, it would be necessary to lie almost on the belly.

The contents of these dropsical sacs are much less tenacious than those of sheaths of the tendons; but not unfrequently these sacs also contain fibrinous bodies, which, on palpation, give a friction-sound, like that made by starch-meal when rubbed between the fingers. In the course of time the sac itself is thickened, the more so the older the disease.

Only the acute cases come under the surgeon's notice. They should be treated as follows: First of all, the patient should be kept quiet; then paint the swelling freely with tincture of iodine. Under this treatment the dropsy generally subsides rapidly; any remaining fluid you may attempt to remove by compression with adhesive plaster or bandages; or you may from the first employ compression with wet bandages, or envelop the knee in wet compresses; mercurial salve and mercurial plaster are also of good service.

Chronic dropsy of the bursa præpatellaris usually causes so little inconvenience that it is generally of long standing before it comes to the surgeon's notice. Most persons scarcely have their movements impaired by the disease; others say that they tire sooner than formerly in the affected limb. The affection is usually limited to one side, but may attack both. It is generally very difficult to cure chronic dropsy of the bursa præpatellaris by the remedies above mentioned. The trouble may be removed by operation. Tapping is no more a radical cure here than in other dropsies, as new fluid collects; for tapping to prove efficacious it should be followed by injection of tincture of iodine. This is free from danger, if the patient subsequently keeps quiet; the result is generally a radical cure. Another treatment is splitting up the sac, which is followed by its suppuration. If the sac be very thick, it is justifiable to extirpate it entirely, which, however, should be done very carefully to avoid injuring the adjacent capsule of the joint. *R. Volkmann* has recommended a plan of treatment which I have often employed with good results, i. e., forced compression; a well-padded, hollow splint of tin or wood is applied to the back of the knee, and the knee is drawn as firmly as possible against it by means of flannel bandages; this compression, which usually causes œdema of the foot, and sometimes severe pain, should be continued several days. Reabsorption results, in two or three days, in small hygromata; in six or eight days, in large old ones. I have seen very good results from this plan, not only in hygroma præpatellare, but also in dropsy of the knee; in dropsy of the sheaths of the tendons it rarely does any good.

[In chronic synovitis, the most efficient form of compression is by the elastic bandage, or by moist sponges kept in place by a bandage. This pressure can only be continued from ten to twenty-five minutes; in the interval an ordinary bandage may be applied. I have seen complete recovery, even after two or three applications of this dressing. Massage, also, which may be applied energetically in such cases, is an excellent remedy; in some respects even preferable to compression. It induces a rapid sero-fibrinous inflammation, which, with the general increase of the blood and lymph circulation, induces absorption of the fluid in the joint. Often massage and compression may be combined, and, in the interval, the limb kept immovable by a splint.

When we resort to injections into the joints, we may try two- to four-per-cent. solutions of carbolic acid, as being less dangerous than iodine, but they also are unreliable. The solution should be brought in contact with the entire surface of the synovial membrane. With this object, the medicament should be introduced to the joint through a large trocar, and motions of flexion and extension made, and the synovial sac distended by the pressure of the injection; the fluid should be changed several times, and finally allowed to escape, or aspirated by depressing the irrigator. Then the opening should be accurately closed, and a *Lister's* dressing and a splint applied. Reaction may be severe, or there may be symptoms of poisoning from absorption of carbolic acid, with symptoms of dangerous collapse. In other cases the effect of the carbolic-acid injection is insufficient, and the fluid reaccumulates.

*Déclat*, a French physician, who published a large book, in 1865, on the internal and external employment of carbolic acid, claims that the dangerous symptoms of poisoning, and the irritant local effects observed from its use, are due to impurities, and to the presence of cresylic acid and other corrosive substances, which he claims may be removed by special modes of preparation.

At present, by careful antiseptic treatment, ganglia may be divided their whole length, the contents evacuated, the cavity washed with a four-per-cent. solution of carbolic acid, drained, and a *Lister's* dressing applied. It is best to continue compression for some time after this operation, by applying a plaster dressing over the forearm and hand, and cutting an opening at the seat of the ganglion, which may be covered by a compress and pressure made by an elastic bandage, while the plaster splint protects the limb from constriction, and keeps it perfectly quiet. After a week the dressing may be removed, the wound being then healed and the fluid not returning. Formerly extirpation of a ganglion was considered a dangerous operation, and correctly so, as one could not know beforehand whether extensive suppuration of the sheaths of the tendons, with all its dangers, would follow; but antisepsis almost certainly prevents this. Hence, by its aid, we may undertake removal of obstinate recurrent ganglia, even if the sac communicates with the sheath of the tendon. Under such circumstances, the opening in the tendon-sheath should be closed with fine catgut, and the limb kept immovable.]

# LECTURE XL.

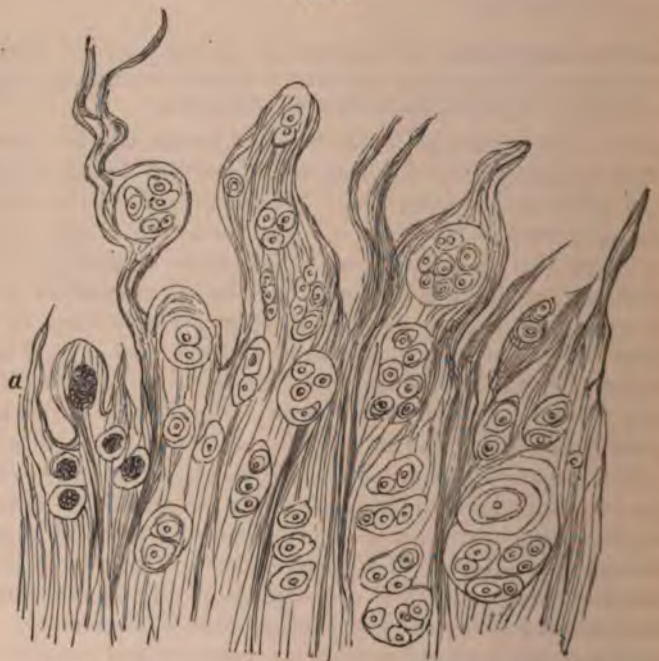
*C.* Chronic Rheumatic Inflammation of the Joints.—Arthritis Deformans.—Malum Coxæ Senile. — Anatomy, Different Forms, Symptoms, Diagnosis, Prognosis, Treatment.—Appendix I.: Loose Bodies in the Joints: 1. Fibrinous Bodies; 2. Cartilaginous and Bony Bodies; Symptomatology, Operations.—Appendix II.: Neuroses of the Joints.

## *C.* CHRONIC RHEUMATIC INFLAMMATION OF THE JOINTS—CHRONIC ARTICULAR RHEUMATISM—ARTHRITE SÈCHE—RHEUMATIC GOUT—ARTHRITIS DEFORMANS—MALUM SENILE COXÆ.

You will be frightened at this crowd of names, which all refer to the same anatomical morbid changes, and you will rightly ask, Why so many names for the same thing? When a disease has received so many designations, it is often a sign that its nature is not correctly understod, or that there have been various views regarding it at different times; but this is not the case here, for the process has always been regarded in the same way, and all observers fully agree in their decisions. It will be best to commence with the anatomy. The disease chiefly affects the cartilage, secondarily the synovial membrane also, as well as the periosteum and bone; in most cases the cartilage is primarily attacked. The changes that we find in the cartilage are as follows: In some places it becomes nodular, then rough on the surface, may be pulled into filaments, and, when the disease is far advanced, it is altogether absent in places, leaving the bone exposed quite smooth and polished. If you examine the cartilage that is broken up into filaments, you will find even microscopically that the intercellular substance, which should be homogeneous, is filamentary. You also find that the cartilage-cavities are enlarged and contain cells, which are dividing up; but these cells are not so small or slightly developed as is customary in cell-formations occurring in inflammations; they are well formed, and sometimes, from a somewhat thickened membrane, are recognizable as new cartilage-cells; the changes progress very slowly, and the newly-formed cells go on to a rather higher grade of histological development than in the above-described forms of inflammation (Fig. 99); the intercellular substance does not soften, as in inflammations generally, but breaks up into filaments; this is a characteristic peculiarity of the disease, but there are also various others. The rough cartilage does not resist the friction of the articular surfaces, but is gradually rubbed through, and is worn down to the bone.

Immediately under the cartilage there is always a layer, even if it be very thin, of compact bony substance; lying next to this are the spongy ends of the epiphyses; after the cartilage is destroyed friction affects this layer, and, as a result of the mechanical irritat new bony substance is formed in this layer; under the point of ir tion the medulla of the spongy substance ossifies to a slight ext The adjacent bones are gradually ground off by the motions in

Fig. 99.



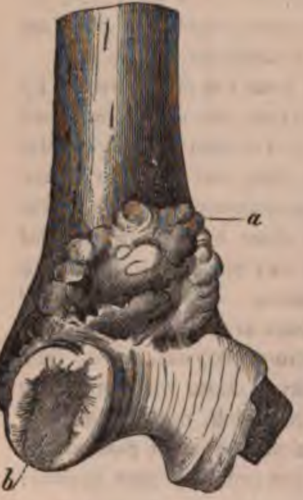
Degeneration of the cartilage in arthritis deformans: *a*, fatty degeneration of the cartilage-cells. Magnified 350 diameters, after *O. Weber*.

joint, but, as the friction constantly causes the formation of new bone, the part ground off usually remains firm and smooth, as the hardening always precedes the atrophy from friction; hence, if the joint remain movable, a considerable portion of the bone may be worn off, and the defective articular surface of the bone may still remain smooth. In the hip, these ground surfaces are at the upper surface of the head of the femur, and in the acetabulum; in the knee, they are on the condyles, etc. In these changes the neck of the femur may be covered with osteophytes in some places, while induration goes on at the smooth surfaces. The neck of the femur may be surrounded by osteophytes, and thus acquire a characteristic shape. This will sometimes come up in very peculiar forms; in one place, atrophy, in another formation of bone, in the same case, alongside of each other in the same bone. The disease not unfrequently begins as nodular proliferation of cartilage, and ends with atrophy of cartilage. I think you are already acquainted with this combination of atrophy and new formation in chronic inflammatory processes; only call to mind caries, the type of ulcerative processes; there we also saw destruction going on at the ulcerated surface, and extensive new formations around it.

The above changes in the cartilage and bone are accompanied by some in the synovial membrane, which, however, do not differ much from those in chronic dropsy of the joint; this contains a slightly-increased amount of synovia, which is cloudy, thin, and mixed with the ground-down particles of cartilage. The membrane itself is thickened, slightly vascular, the elongated tufts alone have more vascular loops in their apices. Parts about the joint may participate in the inflammation—periosteum, tendons, and muscles. These occasionally ossify very slowly, so that the ends of the bones are often covered with bony masses; this bony proliferation is sometimes very extensive. The form of these osteophytes is very different from those with which we are already acquainted; they are flat and roundish, not shaped like pointed stalactites, but look like a fluid which had been poured out and stiffened while flowing; moreover, they are not so porous as other osteophytes, but all the layers are of more compact bony substance. From these peculiarities, which you will at once notice on seeing a series of preparations, the appearance of this variety of articular disease is even exteriorly so characteristic that, on seeing a macerated preparation of the bones, you would at once recognize the disease without knowing any thing of the special case.

In this disease the new formation of bone probably takes such a peculiar form, first, because the process of development is so slow; secondly, because here the ossification is not preceded by any special vascularity, as in osteophytes forming during the union of fractures in caries, necrosis, ostitis, etc.; if a tissue be very vascular when it ossifies, a porous bony substance must be formed, for the more vessels there are the more holes there will be in the bones. But in arthritis deformans the ossification is not preceded by any considerable new formation of vessels, the tissues ossify mostly just as they are; periosteum, tendons, even the capsule, ligaments, and muscles, and all this goes on very slowly; this is why the bone formed is firmer. Sometimes also in the vicinity of the bone in the midst of the subserous cellular tissue detached points of bone form, which for a long time remain isolated round pieces; subsequently they may perhaps unite with the other bony masses; then they look as if glued on, and from the form of the bony growth we may often tell the course of its formation. These periarticular bony formations may cause entire dislocation of the joint and force it into an abnormal, half-luxated position; they

FIG. 100.



Figs. 100 and 102, osteophytes in arthritis deformans. Fig. 100, lower end of the humerus, diminished; *a*, osteophytes; *b*, smoothed end of the bone.

FIG. 101.

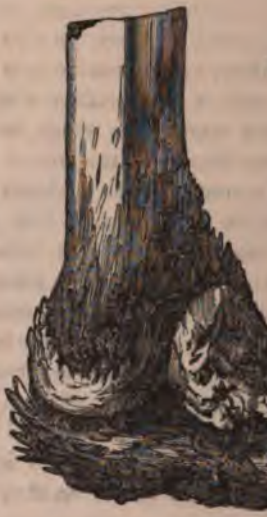


Fig. 101, carious elbow-joint, fungous inflammation of the joints, stalactite-like osteophytes, diminished.

FIG. 102.

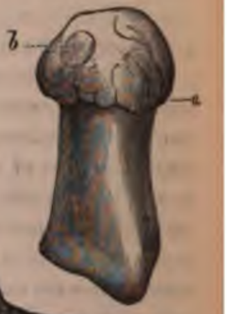


Fig. 102, os metacarpi, I *a* and *b*, as in Fig. 100.

may even render it entirely immovable. Sometimes these osseous formations grow into the joint, loosen from their attachments, and become loose bodies in the joint; of which we shall speak hereafter. Lastly, chronic dropsy may accompany this affection also, and you may readily understand that, from all these concurring circumstances, the joint may become so deformed as justly to deserve the name "arthritis deformans." But, I again repeat, that all these pathological changes never lead to suppuration.

We now come to the clinical appearance of this peculiar disease. According to my experience, I should distinguish three forms of the disease: one, which is usually polyarticular and accompanied by contraction of the muscles; a second, which comes in one joint in young and middle-aged persons; and a third, which only occurs in old age.

1. *Polyarticular chronic rheumatism* (arthrite sèche, rheumatismus nodosus, rheumatic gout) attacks young or middle-aged persons; it is more frequent in women than in men, and in poor than in rich people; badly-nourished, anæmic persons are especially liable to it;

It may originate in acute articular rheumatism or in a gonorrhœal inflammation of the joint; after the termination of the acute or subacute disease of the joints, stiffness, pain, and swelling, remain in some of the joints, most frequently in the knees. But the disease may be chronic from the start, with moderate, unsteady pains in the joints. At first the patients use their limbs very well; but in the course of months and years the mobility gradually decreases; after exertion and catching cold, subacute dropsies of the joint come on, a part of the fluid may be reabsorbed; but the joint always remains somewhat stiffer after every exacerbation, sometimes also it is enlarged. In the morning, when the patient rises, the limbs are so stiff as to be scarcely movable, though, after a few efforts, he gets along better for the rest of the day, but toward evening the joint again becomes painful. Now a new symptom gradually arises; the muscles atrophy, the legs become thinner, and are fixed in a flexed position; the atrophying muscles have great inclination to contract, which is constantly favored by the abnormal position of the joint. Meantime, the general health of the patient remains perfect; his appetite and digestion are good; he grows fat, and only has fever when there is an exacerbation of the joint-trouble. The joint is not very painful on pressure; if it be movable, we may feel and hear friction and grating sounds. This goes on for years. Finally, the patients emaciate greatly, the joints become deformed and stiff, or, as the laity say, "all drawn up;" if the disease be in the hips or knees, they are bed-ridden, but with proper care may live for years; the knee, hip, wrist, ankle, and shoulder joints, are most frequently attacked.

2. *Arthritis deformans* is almost always monarticular, rarely it attacks similar joints on both sides; it occurs in persons otherwise healthy and strong; I have seen it somewhat more frequently in men than in women. This form received its name from the fact that in it the periarticular periosteal formation of bone and the ground surfaces become so extensive that the joint is deformed. I have seen the disease once in the hip, in both knees of the same person, once in the foot and elbow, and twice in the shoulder. Usually there is no assignable cause; in some cases it was preceded by luxations or sprains. These joints are generally painless, stiff, dropsical, and often contain loose bony bodies, and the synovial membrane may be covered with fatty tufts.

3. *Malum coxæ senile.* If the disease attack old people, it is usually somewhat milder than the bad forms of chronic rheumatism. The hip is the chief seat of the disease, hence the name "malum coxæ senile," but it also comes in the shoulder, knees, and elbows, but especially in the fingers and great toes of old people. Its commence-

ment is usually chronic, there is little pain, but much stiffness; more rarely the initial stage is acute; at first, the patients often complain only of stiffness, especially in the morning; after the joint has been used, it grows more movable, the friction is often so marked that the patient calls the physician's attention to it. Attacks with severe pain and slight fever are most common where the fingers are the chief seat of the disease; in the course of years the finger-joints are much deformed. The great toe is dislocated outwardly, and the bony deposits on the head of the first metatarsal bone become very prominent. If the disease develop in the hip, the patients limp slightly; in old persons the bony deposits are generally insignificant; but the thigh is gradually shortened, from the wearing down of the head of the femur and the acetabulum; the muscles atrophy, the hip gradually grows stiff; but this may not take place for years. The disease is much more frequent in men than in women, and thin people are most liable to it. It is rarely accompanied by disease of other organs, particularly the internal ones, but the affection is not unfrequently found in persons predisposed to chalky deposits and abnormal ossifications; rigidity of the arteries, ossification of the ribs and intervertebral cartilages, and anterior spinal ligaments, are often present in patients suffering from malum senile.

The *diagnosis* is easy; after the above description you would not readily mistake the disease. If the affection attack a single joint in a young person, we may at first be doubtful if it is a case of fungous inflammation or of arthritis deformans; but, after further observation, the diagnosis will be easy. In the later stages it might also be mistaken for fungous inflammation, with caries sicca, where we also find atrophy of the muscles and friction in the joint, and which also runs a very chronic course in young and otherwise healthy subjects; but in caries sicca there are never such extensive deposits around the joint, as in arthritis deformans, and, even when of long duration, the latter shows no tendency to suppuration. When the chronic rheumatic articular inflammation occurs on both sides, or attacks several joints at once, and is accompanied by the reflex contraction of the muscles due to irritation of the synovial membrane, the disease cannot be mistaken. Rheumatismus nodosus is often confounded with gout, because the effect of the two diseases on the hands and feet is somewhat similar. But gout is so characterized by its specific attacks, and by the excretion of uric acid, that it should be regarded as a different disease; we have already spoken about this.

The *prognosis* of polyarticular rheumatism is very bad as regards recovery; when it attacks old persons, I consider it entirely incurable. In young patients, by very careful, persistent treatment, the disease may sometimes be arrested at a certain point, and slight improvement be attained; but even this is very difficult, only a few cases are entirely cured. These unfavorable results are due to the anatomical products of this disease; the worn-down cartilage and bone are not replaced, the bony deposits are not reabsorbed, they are too firm and solid; the nutrition of the muscles fails to be excited by the natural motion of the limbs, for they are almost too weak to put in action the stiff limbs. When you have such a patient to treat, arm yourself with patience, and be not surprised if he consults first one then another physician, and finally all the quacks about, and lastly blames you for the origin and extent of his disease.

Of course, even these patients must be treated; the surgeon cannot pick out the curable cases, the incurable and dying also have claims for his aid, and where we cannot aid we should at least try to alleviate and mitigate the disease. Chronic rheumatic inflammation of the joints, by its simultaneous occurrence at different points, shows that it is not due to a local injury, acting on a special joint, but frequently at least to a constitutional cause; the enigmatical rheumatic diathesis is often blamed for the tendency to inflammation of the serous membranes, and exudations in the joints and muscles, hence we employ antirheumatic remedies. The persistent employment of iodide of potash, of colchicum and aconite, of diaphoretics and diuretics, is recommended, although little benefit has been observed from them; but there is nothing else that is better, at least nothing to act specially on the rheumatism. Besides these remedies, and those called for by special peculiarities of the case, warm baths are highly recommended, particularly the indifferent thermal baths: Wildbad in Würtemberg, Wildbad-Gastein, Baden in Zürich, Baden-Baden, Teplitz, Ragaz in St. Gallen; besides these, salt-baths may be given, especially where there is commencing muscular atrophy. Special attention should be paid to the climate of these watering-places, for all of these patients are very sensitive to cold, damp weather. Hot sulphur springs should be tried very carefully, and given up at once if a subacute attack occur after their use. If the patient live in a climate where the winter is cold and damp, he should be sent to winter in Italy, but, for fear of possible cold weather, should only go to places like Nice, Naples, Palermo, etc., where the houses are well built. Damp dwellings should be most carefully shunned. The patient should keep warm, always wear wool next the body, and the affected joints should be wrapped in flannel. Water-cures are much recommended, and show some successful cures; when sensibly used by physicians, and not simply by proprietors of the establishments, they are certainly appropriate, and often prove peculiarly advantageous by hardening the patient, and rendering him less susceptible to external influences, especially to catching cold; moreover, drinking quantities of water, and the wrapping up after the baths, have a diuretic and diaphoretic effect; besides, this mode of treatment has the advantage that patients will follow it out conscientiously and perseveringly, while they soon tire of taking medicines; as is well known, hydropaths soon become enraptured with the system, and are very satisfactory patients even where the treatment is unsuccessful. Hence, if the patient be not too much debilitated, and have no disinclination to the treatment (as sometimes happens), it should be tried, but should be continued at least a year to be of any real benefit. Russian vapor-baths have also been successful in some cases, as have also pine-needle baths. In badly-nourished patients the disease has also been cured by cod-liver oil, quinine, and iron. For local treatment we may rub in various things—the friction is doubtless the most important part of the application; you may use iodine-ointment, simple grease, volatile liniment, etc. Strong derivative remedies are of no use, and even tincture of iodine is only beneficial in subacute attacks, in which cases blisters may also be tried Be careful about applying powerful irritants to the joint; in chronic, torpid cases douches may prove very efficacious; even hot or steam douches and local sulphur-baths have proved beneficial in some cases; but in other cases even the mildest shower-bath, from a foot high, proves too irritating; we cannot always prophesy the effect, the patient should try it carefully under the supervision of the surgeon; as soon as pain is excited, the douche should be stopped, and, after a period of rest, be tried with new precautions; if the pains come on again, and increase, the douches had best be given up.

Should the limbs be kept at rest or moved? For various reasons perfect rest is not desirable: first, because the joint would become stiff, often in a very unfavorable position; secondly, because absolute rest still more increases the atrophy of the muscles. Moderate motion, both passive and active, avoiding the excitation of pain or fatigue, should be made; the patient may make the passive motions with his own hands, or with the very ingenious machine invented by *Bonnet* for this purpose. Lastly, we must add something about muscular atrophy. We attempt to strengthen the muscles by friction, electricity, and regulated movements both active and passive; here curative gymnastics sometimes prove beneficial. But, to be of benefit, any of these methods of treatment must be followed perseveringly.

From this therapeutical review you see we are not poor in remedies that may prove serviceable in chronic rheumatism, but all these modes of treatment are expensive and often unattainable by poor patients, and, as this class are peculiarly liable to the disease, they are very unhappily situated in regard to it. Since dry, warm air, good nourishment, protection from catching cold, and baths, are seldom to be found in the dwellings of the poor, and since these are absolute necessities for the treatment, the prescription of expensive medicines is a pure waste of money. Still, I again repeat, the sooner these patients come under treatment, the more recent the disease, the more you may expect from treatment. You may sometimes arrest the disease. If the malady be already far advanced, its arrest is more difficult, and a cure is rarely to be expected. I believe that most cases of malum coxæ senile are incurable; still, even there the above remedies form the rational treatment. Arthritis deformans monarticularis is incurable. If the joint be much deformed, you may resect it or amputate the limb.

[In the elbow-joint the abnormal mobility caused by smoothing off of the bone is particularly noticeable; in typical cases the bones of the joint may readily be luxated in any direction, and by traction on the forearm may be separated two centimetres; the elbow forms a wabbling joint, in the strictest sense, on which the muscles no longer have any effect; their points of insertion being approximated, the muscles prove too long for normal function.

Joint affections of syphilitic nature occasionally occur under the form of arthritis deformans; these neither belong to the acute period immediately after infection, nor to the results of development of a bony gumma. Their existence is denied by some surgeons, incorrectly I am sure from my own experience. For I have occasionally seen, in children with hereditary syphilis, chronic articular inflammations, especially of the knee-joint, and symmetrical, characterized by moderate collection of fluid and considerable thickening of the ends of the bones, particularly of the condyles of the femur. These have no tendency to suppurate, they are moderately painful, the increased size of the epiphyses impedes motion, especially perfect extension; that these processes are of specific nature is shown by the frequent coincident occurrence of iritis, and by the striking results from antisyphilitic treatment, especially by iodide of potash, without local applications; thickening of the ends of the bone disappears quickly. I know of no post-mortem examinations of such cases. Besides these there are cases of syphilitic disease of the joints, also more particularly of the knee where the cartilage is destroyed and largely replaced by hard, filamentary, brawny connective tissue; the whole synovia is thickened, covered with tufted proliferations, which, however, have not the character of fungous granulations, but consist of firm connective tissue and fat;

the bones remain unchanged. The clinical symptoms are scarcely distinguishable from those of arthritis deformans; moderate pain and impairment of motion only have been observed; there is no suppuration; the disease is seen in young persons (twenty-five to thirty years), and accompanied by other tertiary affections. This disease differs from arthritis deformans by the absence of filamentation and proliferation in the cartilage and the smoothing off of the articular surfaces.]

## APPENDIX I.

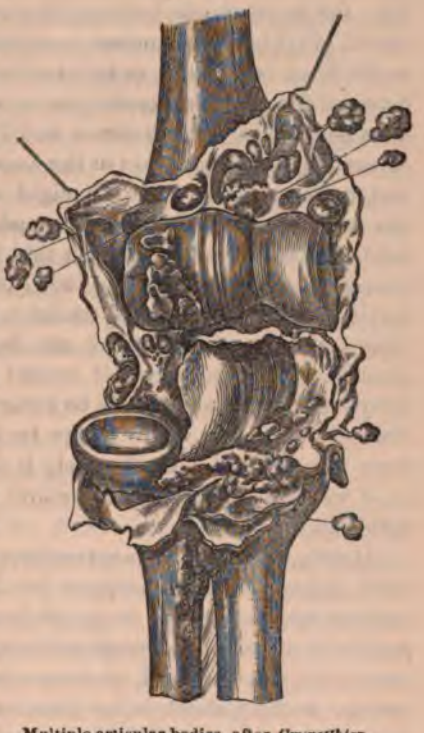
### LOOSE BODIES IN THE JOINTS (MURES ARTICULARES).

By these *loose bodies* in the joints, we mean more or less firm bodies, *forming* in a joint. We exclude foreign bodies entering the joint from without, such as needles, bullets, etc., or detached pieces of bone, lying loose in the joint. There are two varieties of loose bodies: 1. Small, oval bodies, resembling melon-seeds or irregular in shape, which usually form in large numbers, and on microscopical examination are found to consist of fibrine. These form in joints with chronic dropsy, and are deposits from the qualitatively and quantitatively abnormal synovia, just as the analogous bodies are in dropsy of the sheath of the tendons; blood-clots may also possibly serve as a source of origin of such bodies. This form of loose bodies never requires any operation; it is simply an accidental accompaniment of hydrops articulorum chronicus. Occasionally we may predict their presence from finding soft friction when palpating the joint; this does not change the treatment of chronic articular dropsy, and only complicates it in that it renders more difficult the eventual reduction of the joint to its normal size.

2. The other variety of articular bodies is of cartilaginous firmness, generally containing bone-nuclei, sometimes adherent, at others quite loose in the joint. The form is quite varied, being sometimes very odd. The name "joint mouse" (Gelenkmaus) may have arisen from some accidental shape, resembling a mouse. [These loose bodies are usually about the size of a small bean or an almond; in the Vienna Museum is one about as large as the calcaneus, which is attached by a pedicle to the capsule of the knee-joint. One we found in the knee of an old woman was almost as large as the patella, and caused but little suffering.] These bodies are always rounded, but seldom regularly oval or round, being usually nodular or warty; their shape is that of the osteophytes in arthritis defor-

mans. Microscopically they consist of a thin covering of true filamentary or hyaline cartilage, which, from the centre, ossifies, or sometimes only calcifies. As these cartilages are mostly organized, they cannot be regarded as deposits from the synovia; but, even if found quite free, they must formerly have been connected with and have formed in living tissue, and subsequently become detached. The actual process is as follows: These bodies are mostly osteophytes, which have entered the joint from without; *rarely* they form in the apices of the synovial tufts. Even normally there are sometimes cartilage-cells in the tufts; these may proliferate, and thus in the tuft we should have a cartilage-nucleus, a cartilage-tumor, an enchondroma, which subsequently ossifies from the centre. For a time this tumor remains attached to the tuft, but finally it breaks off and then lies loose in the joint. But by far the most frequent form of these articular bodies is from the formation of ossifying cartilages (osteophytes) in the capsule of the joint immediately under the synovial membrane, which may enter the joint and finally tear loose and become free. It is probable that, when once detached and lying free in the joint, these bodies do not grow any more; although it is not impossible that they might derive their nutriment from the synovia. The development of loose bodies is always accompanied by some dropsy of the joint; perhaps the latter is occasionally the primary disease. Loose bodies occur almost exclusively in the knee-joint, and only in adult patients: they are very rare, perhaps the rarest of articular diseases. There is an undoubted connection between the formation of articular cartilages, arthritis deformans, and hydrarthrus. These diseases are of the same class, and from a possibly congenital or developed general diath

Fig. 103.



Multiple articular bodies, after *Cruveilhier*.

esis they form a contrast to the fungous and fungous-suppurative articular inflammations.

The symptoms which may be considered as characteristic of the existence of free bodies in the joint are as follows: The patient has long had moderate dropsy of the knee-joint, and, while walking, suddenly has a severe pain, which prevents his walking for the time being; the knee stands between flexion and extension, and cannot be moved till it has been rubbed in a certain way. This symptom is due to the loose body being caught between the bones forming the joint, between the semilunar cartilages, or in one of the synovial sacs. But, even before this, these patients usually complain for weeks or months of weakness or slight pain in the knee, and, as already stated, examination will generally show a slight amount of dropsy there. From the peculiar mode of occurrence and subsidence of the pain, the patients themselves often suspect that there is a movable body in their knee-joint; not unfrequently they can feel it distinctly, and can, by certain motions of the joint, render it perceptible to the surgeon. In other cases the surgeon does not feel the body till after several examinations, and can move it around in various directions; it often disappears again, and it may be several days or weeks before it again comes in a position where it can be felt. These symptoms only become very evident when the body is detached. While still adherent, or, if too large to be caught as above mentioned, it causes little or no difficulty.

Hence, although the inconveniences of a loose body and of a moderate dropsy of the knee-joint are not always great, and do not increase spontaneously, or go on to suppurative inflammation, and only have occasional subacute inflammation, with serous effusion after some exciting cause, still, in other cases, the pain from the squeezing, and the anxiety about being constantly liable to it, are so great that many patients imperatively demand aid.

The attempt to fix these bodies by adhesive inflammation, induced either by a compressive bandage, tincture of iodine, or blisters, has had little success. The operation consists in the extraction of the foreign body; it is done as follows: The loose body is pressed tightly under the skin, at one side of the joint; the skin over it is then pressed strongly upward, and put still more on the stretch; then cut through the skin and capsule down on to the body, and let the latter spring out, or lift it out with an elevator (perhaps an ear-spoon, as *Fock* has done); instantly close the wound with the finger, extend the leg, let the skin return to its normal position, so that the cut in it lies lower than in the capsule, and the two wounds do not communicate directly; the skin-wound is now to be closed with sutures and plasters, and the limb extended on a splint; a plaster-splint would be very suitable here; one might be made with a large opening and applied even before the operation. According to the symptoms of inflammation that arise, the treatment for traumatic inflammations of the joint is to be instituted. In former times these operations were very unfortunate; they were not unfrequently followed by severe inflammations of the joint, and occasionally the surgeon had to congratulate himself if he saved the patient's life by amputating at the thigh. The modes of operation were often changed; finally that above described, which is the simplest, carried the day. *Fock* performed this operation five times, always with success. The symptoms of inflammation were insignificant, and the patients could usually return to their occupations in a few weeks. As in the extraction of cataract or vesical calculus, much depends on the operation being well performed and without much bleeding or other hinderance. If a loose body causes no inconvenience, we may apply a knee-cap to limit the dropsy and give the joint a certain amount of firmness, so that there shall not be too much motion; this often gives the patient great rest.

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## APPENDIX II.

### NEUROSES OF THE JOINTS.

By neuroses and neuralgias we mean diseases characterized by typical or irregular pains whose causes we cannot find in any change of tissue. We assume that there is a functional disturbance in the nerves without morphological changes. There is no doubt that there are purely functional disturbances, which we call weakness and irritation, in the tissues, and especially in the nerves, where for our senses, even with all modern aids, no morphological or chemical changes are discoverable, either during life or after death. We cannot say if such changes do nevertheless exist; what we cannot perceive with our senses does not exist for us. So that state of the joint where there is pain for which we can find no physical cause is called a "neurosis of the joint." The pains are never typical, i. e., occurring at certain hours of the day in paroxysms, as in neuralgia of the trigeminus, for example. *Brodie* first classed neuroses of the joints as special diseases. *Esmarch*, *Stromeyer*, and *Wernher* have of late paid special attention to these affections; according to them, the group should also include those cases, with slight but still perceptible anatomical changes, where pain and functional disturbance are the chief symptoms, and their severity is out of proportion to the apparent causes of disease. This would place neuroses of the joints among hyperæsthesias with their reflex complications; in short, would class them with hysteria and hypochondria. The cases that I have seen, which from the descriptions of authors were to be placed in this class, I formerly regarded as slight diseases of the joints, whose symptoms were exaggerated in, or even simulated by, hysterical women and girls, or sometimes as commencing and not yet well-defined diseases of joints or bones; and lastly, sometimes as great sensitiveness remaining after the disease had run its course. It is well to have a name for this group of cases, but they are not all to be viewed from the same point or treated in the same way. General medical experience and knowledge of human nature must aid most in the treatment of hysterical patients; the peculiarities and persistence of women in carrying out simulated contractions and spasms is incredible to any one but an experienced physician. Hysteria is really a mental disease, often incurable, or only temporarily curable. For lessening the sensitiveness of joints we may try cold douches, baths, sea-baths, or active use of the joint; the last is especially advised by *Esmarch*. Still, in just such neuroses, which had remained after disease of the joints, I have seen beneficial effects from thermal mud-baths and electrical treatment. Benefit may also be expected from massage.

## LECTURE XLI.

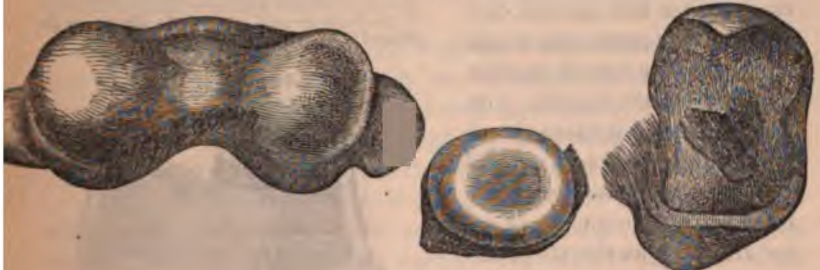
Anchyloses: Varieties, Anatomy, Diagnosis, Treatment; Gradual Forced Extension; Operations with the Knife.

You know that by anchylosis we mean a stiff joint, but I must add that this designation is used only when the process which causes the stiffness of the joint has ceased; that is, when the limitation or total loss of mobility of the joint is the only morbid symptom present. For instance, if during an inflammation of the knee or hip the limb be strongly flexed by involuntary continuous contraction of the muscles, and the joint cannot be extended on account of the pain, although it should be mechanically possible, we do not call it anchylosis of the joint, but articular inflammation with contraction of the muscles.

The causes why a joint cannot be extended after the subsidence of the acute inflammation are partly mechanical hinderances either in the joint or exterior to it, or in parts actually belonging to the joint. A muscle shortened by atrophy and shrinking, a strongly-contracted cicatrix of the skin, especially when on the flexor side of the limb, may greatly impair the normal mobility of the joint; such cases are not meant when we speak briefly of anchylosis; they are termed muscular or cicatricial contraction. Should we term these varieties of limitation of motion anchyloses, it is well to distinguish them as anchyloses from external causes, anchylosis spuria, etc. Now, we have left those cases of stiffness of the joints which are caused by pathological changes of parts actually pertaining to the joint; under this head we have the following cases:

1. Cicatricial adhesions between adjacent surfaces of the joint itself; these may differ greatly in variety and extent; they form after cure of fungous articular inflammations, by adhesion of the proliferating-granulating surfaces; stringlike adhesions are thus formed, like those between the costal and pulmonary pleura, or else there are thick extensive adhesions of the surfaces; along with this state the cartilage may be partly preserved, or it, together with part of the bone, may be destroyed. Generally, these adhesions, like other cicatrices, are formed of connective tissue; in other cases, especially when the joint remains perfectly quiet, this cicatricial tissue ossifies, and the two articular surfaces are united by bony bridges, or else the entire surfaces are completely soldered together (Figs. 104–106).

FIG. 104.



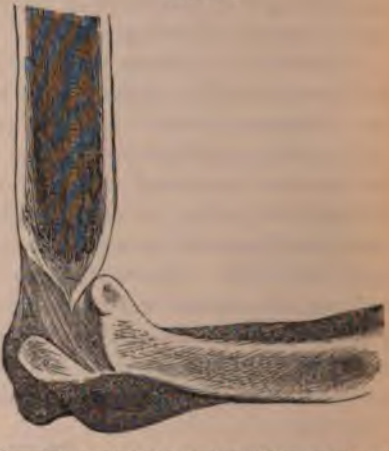
Band-like adhesions in a resected elbow-joint from an adult, almost natural size.

2. Further impediments to mobility are cicatricial shrinkages of the articular capsule, of the accessory ligaments, and even of the semilunar cartilages, which may also be entirely destroyed. These cicatricial contractions occur not only at places where fistulæ have formed, but also when there has been no suppuration, for any tissue that has long been infiltrated, and so more or less softened, subsequently shrinks some, after the process has run its course.

3. A not insignificant impediment to mobility, and one which is the cause of its occasional non-recurrence after extensive fungous inflammations of the joints, lies in the adhesion of the walls of the synovial sacs about the joint, which normally should glide over each other. To render this clear to you, I must touch on the normal conditions of the larger joints in motion. The capsule of the joint is

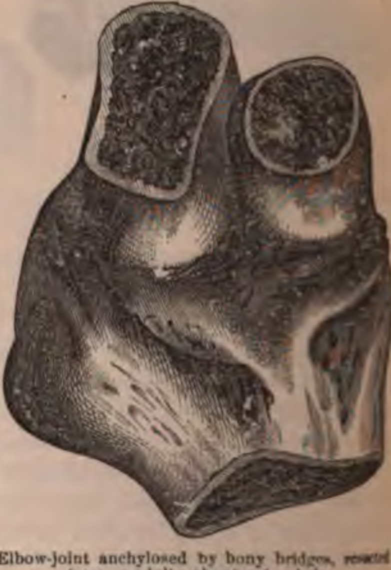
never so elastic as to adapt itself by this means alone to all positions of the joint. If you imagine a humerus lying on the thorax, then at the lower part of the joint the capsule would have to be firmly drawn together, above it would have to be greatly stretched; if you imagine the arm raised as high as possible, the upper part of the capsule would have to be strongly drawn together, and the lower stretched; the articular capsule would have to be as elastic as rubber; this is not the case: on changing the extreme positions of the joint, it contracts little or not at all; it folds up in certain directions; if the position of the joint changes, the fold smooths out, and on the opposite side which was previously smooth another fold forms in the capsule. You here see perpendicular sections of the shoulder-joint, parallel to the anterior surface of the body (seen from the front, after *Henle*) in an elevated position (Fig. 107), hanging by the side (Fig. 108).

FIG. 105.



Complete cicatricial adhesion of the articular surfaces of the elbow-joint of a child, the trochlea of the humerus and part of the olecranon destroyed; section lengthwise, natural size.

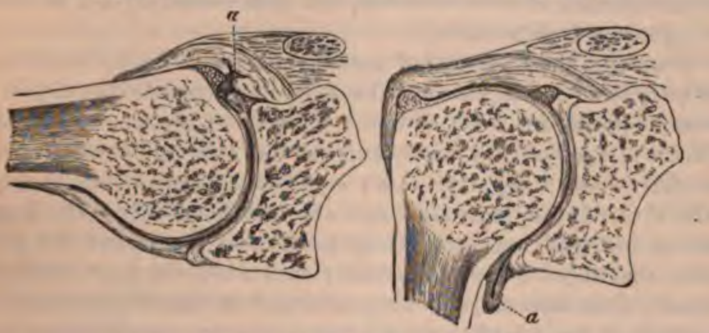
FIG. 106.



Elbow-joint anchylosed by bony bridges, resected from an adult; about natural size.

If the synovial membrane become diseased, the joint usually remains in a certain position, the humerus is generally depressed, the lower part of the synovial sac (Fig. 108, *a*) may suppurate, shrink, and become adherent; then, even if the joint were otherwise healthy, it would be impossible to raise the arm, because the capsule at the lower part of the joint could not unfold. Anchyloses may thus result while the cartilage remains intact; the secretion of synovia ceases, in the course of years the cartilage may degenerate into connective tissue (as in old, immovable luxations), or may even ossify, and the anchylosis will thus become more immovable. Similar circumstances exist in almost all the joints; you will find the best representations of these in *Henle's* anatomy. *R. Volkmann* had previously described this variety of anchylosis, which occurs especially often in young persons after subacute coxitis without suppuration, but with great tension of the muscles, as "cartilaginous anchylosis." The name is well chosen, in so far as in them the cartilage long remains intact.

FIG. 107. FIG. 108.



SECTION OF THE SHOULDER-JOINT, SEEN FROM THE FRONT.
Fig. 95, the capsule folded above, at *a*. Fig. 96, the capsule folded below, at *a*.

4. A further mechanical obstruction may lie in the bony deposits which form in the joint on the articular surfaces of the bones implicated; for instance, if the fossa sigmoidea, anterior or posterior of the lower end of the humerus, fill up with newly-formed bone, neither the processus coronoideus nor anconeus of the ulna can enter it, and in the former case the arm cannot be fully flexed, in the latter it cannot be fully extended. This hinderance is most common in arthritis deformans; it is rare in fungous inflammations of the joint (Fig. 101).

5. Lastly, as a result of caries of the ends of the bones, there may be such loss of substance that the epiphyses will stand obliquely to each other and cannot be brought into position again, because their surfaces are too much changed, and do not fit on each other in the abnormal position (pathological luxation), or cannot be moved at all. Examine Fig. 105 again; as a sequence of the destruction of the trochlea humeri, the ulna is so drawn toward the humerus that, even if some motion were possible, complete flexion could not take place, because the processus coronoideus strikes on the humerus anteriorly, as the fossa sigmoidea is absent. In caries of the knee also the tibia

may be half dislocated outwardly and posteriorly, so that the surfaces which belong together no longer lie in apposition, and in the abnormal position there is no motion at all, or only a slight amount.

Besides these causes of immobility which lie more or less in the joint, there may be external ones, especially the above-mentioned muscular contractions, as well as cicatrices which may become adherent to the muscles, tendons, or bones, and thus materially aid in fixing the joint in a false position.

Generally, the *diagnosis* of anchylosis is not difficult; but it may not be easy to decide which of the above-mentioned factors should be blamed for the deficiency or entire absence of motion. When the stiffness is complete, we readily suppose that there is bony anchylosis, but this is not always the case; very short, strong adhesions, especially if very broad, must also cause absolute immobility. The longer such an anchylosis remains entirely immovable, the greater the probability that there is bony anchylosis; even when the joint is proportionately little diseased, and the greater part of the articular cartilage is normal, if the joint has remained at rest many years (perhaps only as a result of shrinkage of the capsule), complete bony anchylosis will often form gradually; for even a healthy joint will finally become anchylosed if kept immovable for years; motion is an absolute necessity for the continued health of the synovial membrane and cartilage; you may even conclude this to be the case from the fact that all the articulations which are subject to little or no motion (as the intervertebral, pelvic, and sternal), have a very slightly-developed synovial membrane, and are very deficient in cartilage. When the motion of the joint ceases, the secretion of a useful synovia is arrested, the synovial membrane becomes dry, tough, the cartilage becomes filamentary, and the entire beautiful apparatus finally changes to a cicatricial connective tissue which may ossify; then the function of the joint ceases. We have made these statements for the purpose of calling attention to the possibility of deciding, from the duration of an immovable anchylosis, about its firmness. But if the anchylosis be movable, even if very slightly, the synovial membrane is rarely destroyed; part of the cartilage also is usually preserved in such cases. We may be greatly deceived as to the mobility or immobility of anchylosis, if we leave out of consideration the tension of the muscles; frequently, we do not fully comprehend the amount of this mechanical hinderance, till we arrest the muscular contractility by anæsthesia, which must be pushed to the point of total relaxation of the muscles.

Now, what is to be done for these anchyloses? Can we render the stiff joint movable again? In most cases this question can be answered affirmatively. Can we permanently preserve this mobility and restore the normal function even approximately? Unfortunately, this is rarely possible. What shall then be done? What, then, is the use of treatment? This latter question is sometimes a just one, but is not usually so. We have already said that, in inflammations of the joints, the limbs usually assume an abnormal position, a position in which they are very unserviceable; a leg bent at right angles at the knee is a useless, unnecessary burden, hence such limbs were formerly amputated, as the patient could go about better with a good wooden leg than with two crutches. An arm entirely extended at the elbow, or only slightly flexed, is also a very inconvenient member, and very unsuitable for seizing and holding objects, etc. By simply bringing the anchylosed limb into a position where it is relatively most useful, as the knee into the extended position, the arm to a right angle, we may do the patient much good; hence, these operations of straightening or bending anchyloses are very satisfactory. Anchyloses in an inconvenient position were very frequent for a time; they are becoming rarer, and will disappear entirely as soon as universal attention is paid to the principle we urge of placing the joint in the best position for anchylosis, when we are treating acute or chronic inflammations. No surgeon of modern times will have occasion to operate on anchylosis for the improvement of position, in a patient that he himself treated for inflammation of the joint. But there are still many cases that have to be treated in the country under most unfavorable circumstances, where angular anchylosis of the knee or hip results, so that extension of anchylosis is still among the tolerably frequent operations.

Attempts to straighten deformed and stiff limbs are quite old. Even in the surgical writings of physicians of the middle ages we find illustrations and descriptions of machines constructed for this purpose, for the method of relieving the deformities by slow extension with machinery is the older. A large number of apparatus for the various joints have been constructed, by whose aid the extension and flexion of the extremities may be induced by the action of a screw. Now these instruments are chiefly employed in cases where it is thought that, while straightening the joint, we may retain its mobility; but as these cases are very rare, and as they also may be really improved by rapid extension, these machines are much less used. In contradistinction to slow extension of anchyloses, we have the rapid, forcible extension, which is falsely termed *brisement forcé*. Before chloroform was known and employed in these cases, this operation was, on many accounts, objectionable. It was very painful, and not free from danger; it required a great deal of power in the forcible extension of anchylosis for breaking and tearing them up; this was due not only to the obstructions in the joint, but also very greatly to the muscles, which contracted strongly as soon as the pain began. Hence, before trying to extend the anchyloses, it was often necessary to divide the tendons of the tense muscles; this complicated the operation. Moreover, the after-treatment was not correctly understood: the extended limb was bound to a splint, or held firmly by machinery; the consequences were severe inflammation and great swelling; the method did not become popular. *Bouvier* and *Dieffenbach* were almost the only ones who occasionally resorted to it; other surgeons preferred to consider these patients as incurable, or to send them to orthopedists for gradual extension, or, if the patients were poor, to amputate the limb, so that they might have a wooden leg to go about on more securely. So the matter stood till *B. von Langenbeck* in 1846 made the first attempt to extend an anchylosed knee-joint while the patient was anæsthetized. This showed the interesting fact that under anæsthesia the contracted muscles become perfectly relaxed and pliable, and may be stretched like india-rubber; this rendered tenotomy and myotomy unnecessary in this operation. As anæsthesia rendered the operation painless, it could be done more slowly and carefully, and with the aid of the hands alone. The results were so very favorable that this method, which in its new form scarcely deserved any longer the rather brutal name of "brisement forcé," soon became universal, and now it has, perhaps, too much displaced extension by instruments and weights. The method of the operation, the indications, the precautions to be observed, and the after-treatment, were gradually so perfected by *B. von Langenbeck* that this operation may be regarded as one of the safest and simplest in surgery. To prevent your being misled by the name "brisement forcé," and forming too horrible an idea of the operation, I will describe for you the extension of a knee bent at right angles. At first the patient lies on his back, and is gradually anæsthetized so deeply that all the muscles are relaxed, and no reflex movements occur. When this state has been reached, the patient is turned on his belly; one assistant holds the head, another places his arm under the breast of the patient to facilitate respiration; the pulse and breathing are carefully watched, for the operation must be interrupted at once if dangerous symptoms follow the deep anæsthesia. The patient, lying on his face, is to be drawn toward the lower end of the operating-table till the knee comes to the edge of the table, which should be covered by a firmly-stuffed horse-hair cushion. Now an assistant with both hands presses as strongly as possible on the thigh; the operator stands at the outer side of the left (anchylosed) knee, places his left hand in the popliteal space, so as to depress the thigh, and the right on the posterior surface of the leg, corresponding to the posterior surface of the condyles of the tibia, that is, close above the calf, and with his right hand he makes downward pressure on the leg. If the anchylosis be still recent, and not too firm, the leg will gradually give way with a perceptible soft crackling and tearing, and will be straightened by degrees. Should extension not be made so readily, the operator places his hand lower on the leg, about the calf or close below it; but then he should not use so much force as he could above, because he might readily fracture the tibia just below the condyles, especially if the bones were a little soft; the force should here act more in the way of traction or extension. If we do not succeed even by this last means, we should attempt to rupture the adhesions by strong flexion; we seize the leg from the front and try to flex it by slow, regular pressure; by this means the adhesions sometimes rupture more readily than by movements toward extension; after a few of the adhesions have been torn, extension is generally easy. All painful twisting and wrenching is decidedly injurious, and very rarely does any good. When we have made as much extension as we consider prudent for one operation, or, if the leg be fully extended, we turn the patient on the back again, let the assistants press down the thigh by means of *Hueter's* bandages, extend the leg by the foot, and from the foot to within an inch of the perinæum apply a stout plaster-of-Paris dressing, inserting thick layers of wadding at the knee and at the ends of the bandage (below and above, where there is most pressure). But, as the plaster does not always harden before the patient recovers from his anæsthesia, we bind a well-padded hollow splint to the flexor side of the limb, to prevent the knee contracting again; this hollow splint is to be removed after three or four hours; by that time the plaster-dressing is hard enough to resist the contracting muscles. The pain that the patient suffers after recovering from his anæsthesia is not always severe, often it is remarkably slight in proportion to the force employed. The foot sometimes becomes œdematous, if it has not been properly bandaged; but if this has been done, or is done immediately after the operation, there is no further trouble. Should the pain be very severe directly after the operation, we may apply a bladder of ice over the plaster-bandage, and give a quarter of a grain of morphia. After eight or ten days we may allow the patient to gratify his wish of getting up with the bandage on, and going about on crutches, or with sticks. After eight or twelve weeks the anchylosis has healed in its new position. Meanwhile, the patient has thrown aside his crutches, and goes about with a stick, perhaps even without any support, his knee being stiff, but straight; then the bandage may be removed, and the patient regarded as cured.

In the above case we have supposed that an operation succeeded in straightening the knee. But this is not always the case; frequently at the first operation we dare not go so far without risking serious consequences. What circumstances can prevent our completing the operation at one sitting? These are chiefly extensive cicatrices of the skin, which demand very great precautions; cicatrices in the hollow of the knee are especially difficult to deal with, and must be extended gradually; they would be torn if we tried to force the extension. Occasionally, also, the cicatrices surround large vessels and nerves, whose sheaths may have participated in the previous ulceration, and tearing these parts would be a very serious, perhaps fatal complication. Breaking up of any cicatrix may be followed by suppuration, or even mortification; hence we should never stretch cicatrices of the skin to the extreme point to rupture them. Having reached the point where the cicatrices are very tense, we should stop, apply the dressing, and repeat the operation in four to six weeks, and so on till we accomplish our object.

A further circumstance requiring attention is the faulty position of the tibia, that may have resulted from caries of the knee, especially its inclination to luxation backward; it is always difficult, sometimes impossible, to correct this position of the knee, but we succeed best by making the extension very gradually; under such circumstances, forced extension would induce luxation backward — then perfect straightening would be impossible.

You must not expect that the knee will again acquire its beautiful normal shape, even if it be quite straight; this never occurs, but, as we are not called on to go about with naked knees, as the Highlanders do, the shape does not make so much difference, if the knee be only straight and firm enough to walk on. Although joints with tumor albus may be brought into the most serviceable position at almost any time, even when there are fistulæ present, and should be placed in a closed bandage or knee-cap, still, the period when fistulæ have just closed, and the cicatrices are fresh, dense, and tender, is most unfavorable for the extension, for then rupture of the cutaneous cicatrices and new suppuration will be most liable to occur. In such cases I now never resort to sudden straightening under anæsthesia, but always employ extension by weights.

What has here been said in regard to straightening the knee-joint may apply equally to the hip and ankle. Anchyloses of the shoulder and elbow have a totally different functional significance; in them the problem is to restore mobility, and this cannot be obtained by breaking up the anchylosis and applying a plaster-bandage.

If, on straightening a knee, where there have been few adhesions, and the joint is tolerably healthy, we wish to obtain mobility, of course we should not apply the plaster-bandage after the operation, or, at least, should not leave it on long, but we should apply instruments by which motion may be made some time after the extension; this motion should first be tried under anæsthesia, and subsequently repeated daily without the anæsthetic. I shall not deny that cases occur where a tolerable amount of motion may be obtained in this way; but they are rare, and they are either cases where stiffness has remained after fractures through the joint, or after inflammations of very short duration; I could almost believe that, in some of these cases, mobility would have been restored simply by daily use, hence I have no very brilliant anticipations about the results of straightening anchyloses generally. But the mere fact, that we may now almost entirely erase anchylosis from the list of indications for amputation, is a very great triumph over former surgery; but this does not bar the way for further improvements of the new method, or for the attainment of better results.

[Wharton P. Hood (in "Bonesetting," London, 1871) says that partial anchylosis and pain on motion are often due to string-like adhesions. A painful point exists somewhere about the joint; you should press on this with the thumb of the left hand, while steadying the limb above the joint, then with the right hand make sudden flexion and rotation, again extend the limb and let the patient use it at once. Of course this is not to be done if any acute inflammation exists, or in scrofulous subjects.]

Cases occur where the mechanical conditions in the joint are of such a nature that the ends of the bones cannot be brought into any different position. I have already given you the elbow-joint as an example; e. g., the case is one of arthritis deformans, the fossæ at the lower end of the humerus above the trochlea are filled with newly-formed bone; here it is impossible to move the ulna forward or backward; in arthritis deformans similar circumstances occur in other joints, hence the consequent anchyloses cannot be rendered movable, any more than they can after true arthritis, therefore both diseases are usually contraindications to extension of the anchylosis. Lastly, as above stated, the adhesions of the ends of the bones may be bony, there may be anchylosis ossea; it will rarely be possible, indeed, except where there are simply a few osseous bands, to break such anchyloses; in most of these cases the anchylosis will stand firm. What can be done in such cases? There are two ways of altering the position of such joints: by bending the bone above or below the anchylosed joint, or by sawing out a piece from the joint or from the bone. In regard to the first, some surgeons would shrug their shoulders if it were proposed as a method; still, this bending or even fracture of the bone has often been done unintentionally, and has generally turned out well. Several times in extending anchylosis of the knee-joint, once in the hip-joint, without intending it, I made a partial or complete fracture of the bone; the joint remained as before, but above the knee and below the hip the bone bent so as to compensate for the angle at which the joint was anchylosed, and straightening was practically accomplished, although not by rupture of the anchylosis. In all these cases I applied the plaster-bandage; the course was just the same as in simple subcutaneous fractures, the pain was even less than after breaking up anchyloses, and the result was perfectly satisfactory. I cannot see why we should reject this operation of substituting a fracture of the bone for an unsuccessful attempt at straightening the anchylosis, and I should much prefer it to any resection of the knee or hip, where it can be done easily, without great force or hard jerks; I even believe that we should always try to substitute fracture of the femur, if it can be *easily* broken, for resections of the knee at least, no matter how they are done; in other joints resection is of course to be preferred for various reasons. The perfected methods of extension by weights not only enable us to improve the position in most cases of acute and chronic inflammations of the knee and hip joints with unexpected facility, thus avoiding angular deformity; but even in developed anchyloses they prove very effectual, excepting, of course, cases where there is bony anchylosis. Hence extension by weights seems to come more into use and to be an adjuvant to treatment by apparatus as well as by *brisement forcé.*

There are three methods of resecting bony anchylosis: 1. *Rhea Barton's* (published in 1825); in angular anchylosis of the knee, after dividing the soft parts, close above the joint, you saw out from the femur a triangular piece, whose base is upward, and whose angle pointing downward must compensate the angle of the anchylosis (we might also saw this piece out of the anchylosed joint itself); then the limb is straightened, the joint is untouched, the distortion is placed in the thigh, as it is after fracture of the bone. This operation has been done frequently with good results in anchyloses of the hip and knee.

2. We may make a *subcutaneous osteotomy* through the anchylosed joint after *B. von Langenbeck's* method; this operation, which we found to be very useful in fractures that had united obliquely and in rachitis (page 247), has hitherto been little used in bony anchylosis, hence we can give no opinion of it. *Gross* has employed a modified form of it with great benefit; he bores obliquely through the anchylosis in many places, and divides the adhesions with fine chisels.

3. *Total resection of the joint.* I have already stated my opinion about the admissibility of resection for anchylosis of the hip and knee-joints, and would regard it as *ultimum remedium* and *valde anceps;* in the elbow-joint the prospect is rather better; here by resection we may change the anchylosed joint into a movable false one, which is occasionally quite useful, *if all turns out well*, but this is the point on which all depends, and which we cannot always master. Who would risk his life for a stiff elbow? Moreover, in resections for anchylosis of the elbow, the results have not always been very brilliant, either as regards mobility or life, although some cases seemed for a time very successful. So we should not be too free with these resections.

In the shoulder, the circumstances are very peculiar: experience teaches that persons with stiff shoulders can, by constant use, make their shoulder-blades so movable that the stiffness of the shoulder causes comparatively little inconvenience; in such a case it would be folly to operate.

Patients with caries of the wrist are usually so glad, when, after years of suffering, the disease at length recovers, that they do not complain of their stiff hand; nevertheless, successful resections of anchylosed wrists have been recently made by *Rose;* it is true, the final results of these operations are not yet fully known. In the foot there would be no question about resection for anchylosis in a bad position; usually defect of the ankle-bones is the chief cause of deformities of the foot after inflammation of the joint. It will depend on the individual case whether the foot is useful, whether a correction of position be possible, or if a good stump be preferable.

# CHAPTER XVIII.

## *CONGENITAL DEFORMITIES OF THE JOINTS, DUE TO MUSCULAR AND NERVOUS AFFECTIONS AND CICATRICIAL CONTRACTIONS—LOXARTHROSES.*[1]

## LECTURE XLII.

**I. Deformities of Intra-uterine Origin due to Disturbances of Development of the Joint.—II. Deformities occurring only in Children and Young Persons, caused by Impaired Growth of the Joint.—III. Deformities from Contractions or Paralysis of Single Muscles or Groups of Muscles.—IV. Limitation of Movement in the Joints from Contraction of Fasciæ and Ligaments.—V. Cicatricial Contractions.—*Treatment:* Extension by Apparatus.—Straightening during Anæsthesia.—Compression.—Tenotomy and Myotomy.—Division of the Fasciæ and Articular Ligaments.—Gymnastics and Electricity.—Artificial Muscles.—Supporting Apparatus.**

GENTLEMEN: To day we have to speak of those deformities not resulting from primary disease of the joint, but leading to abnormal mechanical conditions, if the articular surfaces from various causes assume abnormal forms, or if while the form remains normal the movements be impaired in some direction by obstacles due to abnormal states of the muscles, fasciæ, tendons, or skin. Most of the cases are of stiffness, deformity, or limitation of motion of the joint, exterior to the synovial membrane. In this section I follow chiefly the division of *Volkmann*, whose extraordinary work on this subject, published in *Billroth* and *Von Pitha's* Archives, I cannot too strongly urge your studying.

### I. DEFORMITIES OF EMBRYONAL ORIGIN, DUE TO DISTURBED DEVELOPMENT OF THE JOINT.

These distortions are always congenital; they are much the most frequent in the foot, especially as *club-foot*, *pes varus seu equino-varus.* Although we may, and formerly did, term all distortions where the foot was drawn together into a "clump" as club-foot, we now generally mean by this term only the forms where the inner border of the foot is raised, while the plantar surface is usually flexed, and in children it cannot be brought into the normal position, unless with the greatest difficulty. If children born with such feet (both feet are usually affected) learn to walk, they step on the outer side of the foot; this rolls more and more inward, becomes flat, the hollow of the foot is contracted, the middle and anterior part of the foot are not well developed, the joints become anchylosed and the feet become misshapen clubs; the outer part of the back of the foot is the part walked on, and at that point a thick callosity forms with a mucous bursa under it; as the foot is not moved, the muscles of the leg atrophy, so that little besides skin and bone is left; this causes the resemblance to a horse's hoof. Various grades of club-foot have been distinguished, from the trifling deformity just after birth to that just described. It is to be remarked that the higher grades of club-foot result from walking; if the patient never got on his feet, the congenital deformity would probably change little, if any.

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[1] From λοζος, oblique, ἄρθρον, member, joint.

The most varied hypotheses have been advanced as to the causes of congenital club-foot. The typical form of this congenital deformity appears to indicate that it depends on disturbance of a typical development of the lower extremities; for if fœtal disease, disturbance of an irritative nature, or abnormal pressure in the uterus, were at fault, cases would probably differ, as we shall see hereafter. The following views, recently published, seem to me very important in the explanation of this deformity. *Eschricht* has shown that at the commencement of their development the lower extremities lie with their backs against the abdomen, the hollows of the knees being against the belly; so during the earlier months the legs must rotate on their axes, and the toes, which pointed backward, must point in the opposite direction. If the embryonic extremities lie so close as to appear united under a common skin, or be really united, the above-mentioned rotation of the limbs cannot occur, and in this deformity (*siren*) the feet are turned directly backward. This rotation on the axis, which was arrested in the above case, does not take place fully in club-foot, the rotation in the foot is not fully accomplished. According to this, congenital club-foot would come among cases of obstructed development; about its cause we know as little as we do of other deformities of the same class. The abnormal forms observed by *Hueter*, especially the obliqueness of the ankle-bones, unsuitable length of the muscles, among which shortness of the gastrocnemius is the most conspicuous and longest known, must be regarded as consequences of this faulty direction of the foot *in utero*, which is subsequently increased. This explanation, based on accurate observation, is so much more satisfactory than the previous hypothetical explanations, which mostly referred the affection to fœtal myelitis, with consecutive paralysis and contraction, that the latter scarcely deserve mention except for their historical interest.

Some other congenital deformities of the feet are proved to be due to abnormal positions, and especially to abnormal pressure. *Volkmann* has collected some very interesting observations on this point; but these cases differ among themselves, showing that there was something accidental in their occurrence. In still other cases large portions of bone remained undeveloped, e. g., the lower end of the tibia, fibula, or radius, or the whole radius (manus vara).

In the spinal column, lateral halves of the vertebræ sometimes do not develop fully, or superfluous pieces may be formed, causing lateral curvature (scoliosis); but these congenital cases of curvature are very rare; the Vienna collection has a few of these rare specimens. Lastly, we must here mention the faulty development of the sterno-cleido-mastoid muscle, which is not rarely congenital and is quite typical; the vertebræ remain normal, so far as is known; we know nothing of the causes of this deformity; the hypotheses I have seen seem to me scarcely probable.

## II. DEFORMITIES DEVELOPING ONLY IN CHILDREN AND YOUNG PERSONS, CAUSED BY DISTURBED GROWTH OF THE JOINTS.

All conditions of the body, such as standing, walking, sitting, etc., depend on the forms of the joints and their ligaments, and on muscular action. The effect of the latter on all our positions, even in the way we lie, you may best perceive by trying to give a certain position to a cadaver, from which the *rigor mortis* has passed away; you would then see that we rarely take the natural positions given by the form of the joints and ligaments, but generally aid them by the muscles. Persons whose muscles tire easily, from weakness, exhaustion from disease, or lack of exercise, in assuming any position will of course seek the one most natural, and requiring least muscular action. The articular pressure due to muscular action is always evenly distributed over the whole articular surface, and, when this action diminishes or ceases, individual parts of the ends of the bones forming the joint have to bear all the pressure. If the bones were fully developed and firm, this unusual burden would have no further results, if it were of short duration. But when growing bones, which are still soft, and will remain so for a time till their forms are fully developed, are repeatedly and for a long time exposed to pressure, acting on the same point, the form of the articular surface and of the articular ligaments gradually changes; the bones also sometimes fall into a state of pathological softening, accompanied by pain and the disturbances of growth in the ends of the bones caused by the abnormal burden, increase rapidly; there is a corresponding change in the ligaments and muscles, and the changes begun here, react on the form and development of the entire skeleton. The most important examples of this are *scoliosis*, *genu valgum*, and *pes planus.*

By "scoliosis" (from σκολιός, curved) we mean the state of the spinal column, where it is constantly bent to one side, and where this curvature has become permanent. As already mentioned, such a position may arise from abnormal formation of the vertebræ; it may also be due to enormous distention of one side of the thorax from pleuritic effusion, or to collapse of one side of the chest from reabsorption or evacuation of such effusion, or lastly to fixation of the pelvis in an oblique position, either from apparent or real shortening of a leg after a joint or bone disease, or other cause. All these are relatively rarely the causes of the scolioses of which we are here treating; these usually occur in young girls shortly before puberty. These curvatures have a typical form; as a rule the lumbar portion of the spinal column is convex to the left, and the upper dorsal portion is convex to the right. It is a matter of dispute whether the lower or upper curvature comes first; whether the first is the primary and the latter secondary or compensatory, or the reverse; as a rule we find both curvatures from the start, they probably develop about the same time. If the faulty position remain unobserved and without treatment, and the unfavorable conditions continually increase, the right scapula is elevated (the first marked symptom), and as the vertebræ gradually rotate the deformity constantly increases, the upper part of the spine projects as a gibbosity, the position of the head changes correspondingly, the thorax is displaced, in short a hump-back is developed. From anatomical reasons, which have been carefully traced by *H. Meyer*, the protrusions of the spine posteriorly (cyphosis, from κυφος, gibbosity) always accompany high grades of curvature, so these deformities are also called "cypho-scolioses." Most old persons with humps belong to this class; patients with caries of the vertebræ rarely attain old age; hence, we only see the so-called Pott's curvature caused by caries of the vertebræ, in children and very young persons. The chief cause of scoliosis is weakness of the spinal muscles; as long as feeble children are left entirely to themselves, and can lie down, sit, walk, or run, as they wish, and as long as they feel like it, scoliosis rarely develops; but when they are made to occupy certain tiresome positions for hours, as in writing, reading, sewing, playing the piano, etc., they will seek positions where the muscles for keeping the body erect are used the least. These positions become customary or habitual. When the children are sitting, even without occupation, and politeness forbids their lounging, they support themselves with one hand on the seat; if they stand, they lean so that the body does not need to be supported; they usually stand on one leg to rest the other. If the curvature of the spinal column has existed for months or years, the centre of gravity of the back and head changes more rapidly, and the deformity progresses more quickly. At first, the intervertebral cartilages are merely compressed on one side, then they become relaxed on the other side, grow thicker, and the bodies of the vertebræ are compressed more and more, till, from being cylindrical, they become conical. This compression sometimes also leads to inflammatory new formations, to deposits of osteophytes, occasionally even to ossification of the ligaments.

*Genu valgum*, or *baker's leg*, is a deformity of the knee-joint, where its shape is such that the leg forms, with the thigh, an obtuse angle externally; if these patients lie on the back, placing the inner sides of the knees together, the feet will stand far apart; to place the inner borders of the feet together they must cross the knees. This deformity occurs most frequently in young males, who during the whole day have to move their bodies and arms while standing, and at the same time have to bend their knees; bakers', locksmiths', and cabinet-makers' apprentices are especially predisposed to this affection, which is very painful when it is excessive or increasing rapidly. The external condyle is strongly compressed, the internal lateral ligament much stretched, the external lateral ligament contracted, and the biceps muscle is shortened and becomes tense.

*Flat-foot*, *pes planus*, is a deformity of the foot, frequently affecting young girls as well as boys before the age of puberty, when they have to stand much. The bones, which form an arch at the inner margin of the foot, sink down so that the sole of the foot becomes perfectly flat, or even convex downward; then the outer border of the foot is elevated (pes valgus), and the peronei muscles are shortened, their points of insertion being approximated. This deformity of the foot is very frequent; it often follows genu valgum, but oftener is independent; sometimes it comes on very rapidly and with severe pain.

While I consider the above-mentioned continued pressure on growing bones as the essential cause of scoliosis, genu valgum, and pes planus, still we cannot fail to notice that only a few of the persons subjected to these injurious influences are affected with the above deformities, so that we are naturally led to suppose that besides the muscular weakness there must be a special weakness of the osseous system, a softness of the bones; indeed, I cannot help thinking that there is a slight amount of rachitis. *Lorinsen* and some other authors claim that the above cause is very prominent in the etiology of curvature of the spine. *Hueter*, *Henke*, and other authors, claim that in all these deformities the articular surfaces grow obliquely and uneven; this certainly has much to do with the increase of the disease, but can scarcely be recognized as a cause. The results of recent investigations render it improbable that (idiopathic) contraction and relaxation of the articular ligaments cause these deformities, as I was formerly inclined to believe, although, from the displacement and deformity of articular surfaces of the bones, they must occur.

## III. DEFORMITIES DUE TO CONTRACTIONS OR PARALYSES OF SINGLE MUSCLES OR GROUPS OF MUSCLES.

This class of cases is very numerous. Acute inflammations in muscular substance, or in the immediate vicinity of muscles under tense fascias, may cause contractions simply by rendering the stretching of the inflamed muscle very painful. It is very common in deep abscesses of the neck to find the head inclined to the affected side, so that the patient is entirely unable to straighten it; and this can only be done under anæsthesia, when it is readily accomplished. I once saw a foot fixed in the position of pes equinus by an abscess in the muscles of the calf of the leg. Acute inflammation of the psoas muscle (psoitis and peri-psoitis) often causes the hip-joint to be flexed at an acute angle. When the pus is evacuated these contractions diminish, and often gradually disappear entirely; but sometimes the cicatrix is so large that it continues the contraction, which is afterward removed with difficulty.

Secondly, direct nervous irritation from disease of the nervous centres may cause permanent contractions; when these cases start from the brain, they offer very little chance for treatment. In caries of the spinal column and transfer of the inflammation to the anterior roots of the spinal nerves, muscular contractions and paralysis of the limbs sometimes occur simultaneously; in one such case I saw a nearly complete cure occur spontaneously.

Reflex paralysis may also occur. I have seen such cases where the thigh, hand, and foot, were affected, particularly in young women; in some cases these contractions were induced by falls on the parts, in others by irritation of the genital system (hysterical contractions). These cases relax during sleep and anæsthesia.

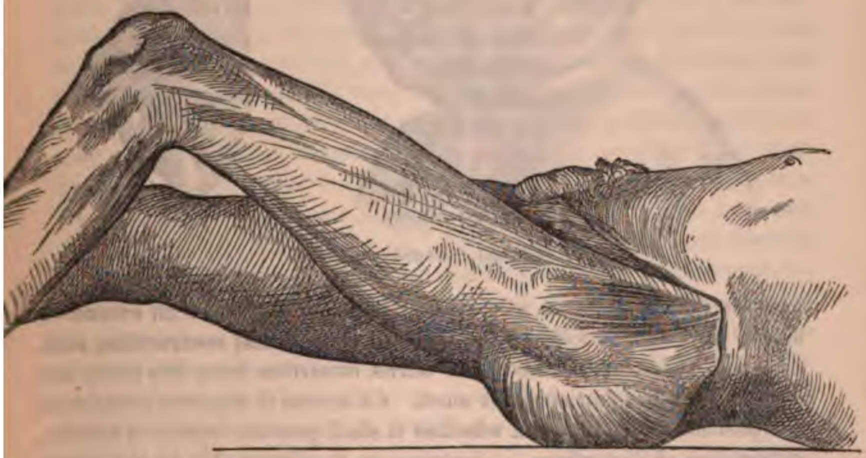
Lastly, we come to the most frequent of all of these groups, the so-called *paralytic contractions*, such as occur after partial or total paralysis from meningitis and encephalitis, especially in children. These contractions occur on one or both sides, chiefly in the lower extremities. From its mechanical construction a completely paralyzed leg hangs and lies with the foot extended, and turned somewhat inward; of this you may convince yourself by examining any cadaver which is not rigid. If the foot be not purposely brought out of this position, it becomes gradually fixed there, partly by the ligaments on the back of the foot, the muscles of the calf, tendo achillis, and superjacent fasciæ atrophying, partly by the slower growth of these parts; gradually also the articular surfaces and the form of the bones change as a result of unequal pressure, as previously explained, and it becomes more difficult and at last impossible to bring the foot into a right-angled position; in attempting to effect this the resistance from the muscles and tendons is most readily perceived, hence the opinion that the gastrocnemius muscle and the tendo achillis were contracted, even in cases where it was just as much paralyzed as the other muscles of the leg. Then it was thought that only the extensor muscles were fully paralyzed, while the antagonists preserved some innervation, so that they alone acted on the foot and attained a relative preponderance. Thus arose the theory of antagonistic contractions taught especially by *Delpech*; which was sustained chiefly by those cases where there was really an unequal distribution of paresis and paralysis of the different groups of muscles. It was *Hueter* who first called attention to the fact that it was chiefly the continued position assumed by the paralyzed limb, from its weight, that induced the contractions, and that these so-called antagonistic contractions were not at all muscular actions, but, as in congenital club-foot, were due to atrophy and lack of growth. After investigating this view, I must entirely agree with it. I had often met cases where the theory of antagonistic contractions seemed doubtful; as in one case where at the battle of Sadowa a soldier was shot through the right forearm and had the radial nerve torn; four years subsequently there was total paralysis of all the parts supplied by that nerve, but not a sign of antagonistic contraction. If we carry our investigations to paralyzed limbs, we find that, in cases where the patients sit all day with the leg and thigh flexed, contractions take place at the knee and hip, but, if the patients with partly paralyzed limbs still have strength enough to move about with support, the movements of the joints continue up to a certain point. This also you may best see on a cadaver that is not rigid; the foot placed on the ground, with the body resting on it, turns outward (pes plano-valgus paralyticus), the knee bends forward and outward (genu antecurvatum), while at the hip the body falls forward till it is supported by the sound leg, crutches, or cane

Thus, from the weight of the body, the limbs assume positions which gradually become permanent (*Volkmann*), and in young persons have a decided influence on the forms of the articular surfaces. All these conditions may be most naturally explained on mechanical principles, while formerly the most complicated theories were based on very slight grounds, when any explanation was attempted.

## IV. LIMITATIONS OF MOTION IN JOINTS CAUSED BY SHORTENING OF FASCIÆ AND LIGAMENTS.

Any long-continued fixed position of a joint, even if not due to the above-described diseases of muscles and nerves, may lead to shortening of the fasciæ. A man who kept his left leg and thigh flexed for a year and a half, on account of suppuration of the inguinal glands, was brought to our clinic after the bubo had healed, because he could not extend the leg. This is particularly true of the fascia lata, which from a few months of quiet may become so rigid that it is sometimes impossible to extend it again. After coxitis has run its course, when the joint has become perfectly healthy, this contraction of the fascia may prove a permanent obstruction to complete extension, so that such patients may occasionally limp for life; which is another important reason for paying special attention to the position of the limbs in inflammation of the joints.

FIG. 109.



Contraction of the fascia lata from coxitis, after *Froriep*.

## V. DEFORMITIES CAUSED BY CICATRICES.

We have already spoken frequently of the contraction of cicatrices; it results from the inflammatory new formation in the wound gradually giving off water, as the original gelatinous formation by degrees atrophies to dry connective tissue, and contracts like any body that is drying up. The larger the surface of the cicatrix, the stronger will be the contraction in all directions; all wounds with extensive loss of skin will be followed by extensive cicatricial contraction, and, as this is generally greatest after burns, cicatrices from this cause are usually the ones that contract most. Of course it depends greatly on the position of the cicatrix whether it shall produce injurious results, deformities or distortions. Cicatrices on the flexor side of the joint, when they extend far longitudinally, may prevent full extension of the limb. Extensive cicatrices in the neck induce distortion and fixation of the head to the injured side; those on the cheek may distort the mouth or lower eyelid; on the back of the hand or foot, or about the finger-joints, they may render the finger immovable, or partially so.

FIG. 110.

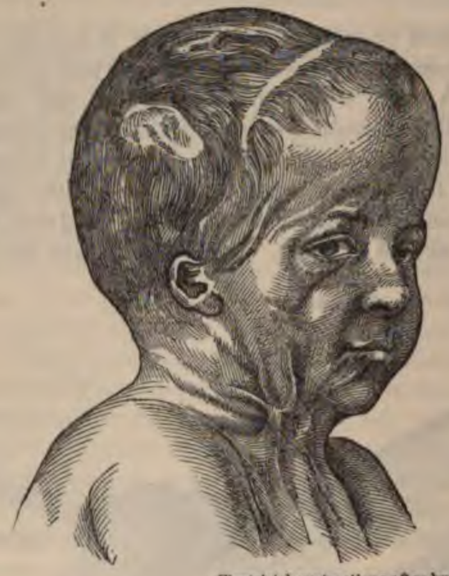
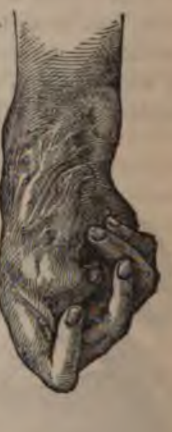


FIG. 111.



Cicatricial contractions after burns.

But cicatrices of the deeper parts, as of the muscles and tendons, may, of course, also cause deformities; as necrosis readily follows injury of a tendon, and cicatricial tissue replaces the tendon, such a part as a finger, when injured, becomes crooked and stiff.

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Although, in what has just been said, we have spoken chiefly of the etiology of deformities, still the diagnosis is included there; and it is unnecessary to pursue this point further. Of course the *prognosis* depends entirely on the possibility of removing the causes, and the treatment also varies greatly with the latter.

To remove contractions, the most natural thing is to stretch the parts; we may try this by having the contracted limb stretched a few times daily. But this so-called *manipulation*, which is very efficacious, requires much strength and patience; hence it seems better to make this extension by the regular action of a machine. The extending machines now used depend on the combined action of the screw and cog-wheel, a mechanism that has been employed in surgical instruments from the most ancient times; the machines may be variously constructed, but must be light, firm, and well padded; they should never press too hard, and be made to retain any position; such machines are most readily made for the knee and elbow; in the shoulder and hip it is difficult to fix the scapula and pelvis. Extension may be made under anæsthetics, to hasten the progress; but then avoid using too much force, and especially bear in mind that cicatricially-contracted muscles are less distensible than normal ones, and can only be stretched gradually. Mechanical extension can scarcely be applied to those muscular contractions depending on neuroses, or, at most, it can only be used as an adjuvant; the chief treatment must be directed to the nervous affection that has caused the muscular contraction. Not unfrequently these contractions entirely disappear under chloroform, especially when of a reflex character, in the same way that they subside spontaneously in acute articular inflammations, as soon as the patient is narcotized; the flexed knee, for instance, may then be extended without the least force. According to *Remak*, many contractions disappear under the use of the constant current of electricity; as many excellent men are now engaged studying the constant current, it is to be hoped that the mystery, which has until lately shrouded this subject, may disappear before clear criticism. Treatment by apparatus (*orthopedy*) is particularly used in contractions of ligaments and fasciæ. Contractions from cicatrices may be improved, but rarely entirely cured, by stretching the cicatrix; a more potent remedy here is *continued pressure*, made by adhesive plaster, bandages, or compresses, applied to suit each case. The atrophy of the cicatrix, which occurs spontaneously, in the course of years is much promoted by this treatment. Distention is combined with compression in the treatment of ring-shaped cicatricial contractions of canals, so-called strictures, such as occur chiefly in the urethra and œsophagus, by the introduction of elastic sounds (called bougies because they were formerly made of wax) of gradually-increasing thickness.

The orthopedic treatment previously mentioned does not always succeed, or at least is often very slow, hence even in the middle ages the tendons of the contracted muscles or the muscles themselves were divided; this operation is called "tenotomy," or "myotomy;" the former is far the more frequent. Formerly the operation was done by simply incising the skin down to the tendon, then dividing the latter, and letting the wound heal by suppuration; the results were not very brilliant: the suppuration was sometimes very extensive, thick cicatrices formed, which could only be slowly stretched. This operation was first made really serviceable by *Stromeyer*, who taught us to divide tendons subcutaneously, a method which *Dieffenbach* introduced extensively into practice, and which is now exclusively used. I shall first describe this operation briefly before passing to its results. Let us take, as an illustration, tenotomy of the tendo Achillis, which is the most frequent. For this operation you may best employ *Dieffenbach's* tenotome, a slightly-curved, pointed, narrow knife. The patient lies on the belly, an assistant holds his leg firmly at the calf; with your left hand you seize the club-foot; with your right hand introduce the knife, flatwise, by the side of the tendon under the skin, and over the tendon, till you have passed beyond the tendon, without, however, perforating the skin a second time; now turn the edge of the knife toward the tendon and divide the latter—when so doing you will hear a crackling sound; as the division is completed, you will feel with the left hand that the foot is more movable; you now carefully draw out the knife. Only the point of entrance of the knife is visible externally, the tendon has been divided subcutaneously. This method of subcutaneous *tenotomy from without inward* is easier for beginners, because in it there is no danger of dividing the skin more than is necessary. *Tenotomy from within outward* is more elegant and better suited for some cases. The foot is held as above, and the knife is entered the same way, but it is then passed under the tendon and the cutting edge turned toward the tendon; the thumb of the right hand should be placed over the point of the knife to feel it and prevent passing it through the skin; we then press on the knife and draw it from within outward through the tendon; being careful not to let it cut through the skin when the jerk occurs that accompanies the completion of the division. This method seems more difficult than it is, but, like any operation, it requires practice on the cadaver. When the tenotomy is completed, there is usually but little bleeding from the puncture, though sometimes there may be considerable, as in some persons a tolerably large branch of the posterior tibial artery runs alongside of the tendon, and is divided with it. If the bleeding be slight, a piece of ichthyocolla-plaster may be placed over the puncture, and rendered firmer by collodium; if the hæmorrhage be more profuse, the puncture should be covered with a small compress, and the foot bandaged as high as the calf; the bleeding then ceases. This dressing should be replaced by plaster after twenty-four hours. The healing is almost always by first intention; the puncture is closed in three or four days. But there may be suppuration; then the wounded part grows red, swollen, sensitive; blood mixed with pus flows from the wound, an abscess often forms on the opposite side; this must be opened, and, although this suppuration is not dangerous to life, it may continue two or three weeks, and much impair the results of the operation, for it is a long time before the resulting thick cicatrix is suited for extension. Immediately after the tenotomy, at the point of division you may feel a hollow, as the muscle contracts after division of the tendon; this hollow disappears in the course of twenty-four hours, and for a few days it is even replaced by a swelling; the latter gradually subsides, and in fourteen days at most, after a normally-healed tenotomy, the tendon appears perfectly restored. The course of this healing has been carefully studied experimentally; formerly it was supposed there was something very peculiar about it; I have often made these experiments on animals, and find that healing takes place as it usually does, and most resembles that process in nerves and bones. When the tendon is divided, and the muscle contracts, there would be an empty space at the point of division if the external atmospheric pressure did not at once press the surrounding cellular tissue into the space between the ends of the tendon; the space is thus filled up; as in any wound, this tissue is infiltrated with plastic matter and serum, and becomes very vascular; the cellular tissue around the ends of the tendon is metamorphosed in the same way, and the latter are surrounded and united by the inflammatory new formation developed from the adjacent cellular tissue, just as the fragments of bone are by the external callus (which, however, here presses

FIG. 112.

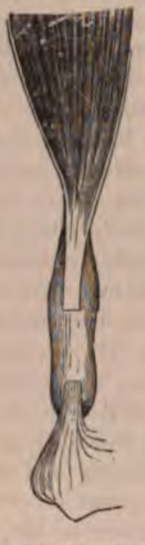

Diagram of a subcutaneously-divided tendon, on the fourth day.

*between* the ends of the tendons also; an internal callus cannot develop in tendons, as they have no medullary cavity). In this stage (about the fourth day), the picture is somewhat as in Fig. 112.

This provisional union soon becomes firm, as the inflammatory new formation is metamorphosed to connective tissue; meantime, some neoplastic tissue has developed in the stumps of the tendon, which combines with the intermediate substance. The entire newly-formed intermediate mass gradually contracts strongly, becomes very firm, so that it assumes exactly the character of tendinous tissue; the tendon is thus entirely regenerated. It is true this does not always go on as rapidly as we have here described, but (as also occurs in fractures) is not unfrequently interfered with by a large extravasation of blood between the ends of the tendon; this is enclosed by the inflammatory new formation, becomes only partially organized, but must be mostly reabsorbed before there can be complete regeneration of the tendon. Extensive extravasations of blood may interfere with the regular course of healing, not only by their size and the time required for their absorption, but by occasionally putrefying and suppurating. The operation and course of healing in myotomy are about the same as have just been described.

You have just heard that the tendon is entirely regenerated, and the cicatricial intermediate substance contracts strongly, that is, it shortens, and you will justly wonder why, knowing these facts, the operation is still done, as the tendon is not thereby much elongated. To this I answer that tenotomy of itself is of no use, or, at least, does little good, but that the tendinous cicatrix may be much more readily stretched than the tendon of the contracted muscle or the muscle itself; tenotomy only proves useful from the orthopedic after-treatment; it greatly aids the cure, and often it alone renders it possible, when the contracted muscles, fasciæ, or ligaments, resist all efforts at extension. Hence we should not await complete cicatricial contraction of the divided tendon, but must stretch the young cicatrix; the orthopedic treatment may begin ten or twelve days after division of the tendon in club-foot, either by extension, manipulations, and apparatus, or by straightening the foot and applying a plaster dressing. Favorable results were first rendered possible by *subcutaneous* tenotomy; then the healing goes on rapidly, and a distensible cicatrix forms; if the wound suppurates a long time, and the skin is also affected, the brittle cicatrix probably may not become distensible for six or eight weeks, for sooner it might tear and begin to suppurate again. Of course every club-foot, especially of the lower grades, does not require tenotomy; but it is just as certain that in high grades of this deformity tenotomy favors the cure. From what has been said, you will see that the indications for tenotomy are often the same as those for orthopedic treatment; this is not absolutely the case; the indications for tenotomy are sometimes more limited, sometimes more general. We *may* divide any tense tendon subcutaneously; but whether this will do any good is another question. We cannot here speak of all possible cases, but I will mention the tendons most frequently divided: in the neck, the two portions of the sterno-cleido-mastoid muscle, at their insertions on the clavicle and sternum; tenotomy is rarely done in the arm; I warn you against this operation in the fingers and toes; *all tendons with fully-developed sheaths are unsuited for tenotomy;* from anatomical reasons, that you may readily perceive, healing would not occur so simply as in tendons surrounded by loose cellular tissue; there is usually suppuration, frequently with bad results, or else the ends of the tendon remain ununited. In the thigh, after coxitis, the contracted adductor muscle may be divided at its point of origin, if its contraction cannot be overcome during anæsthesia; the same is true of the biceps femoris, semitendinosus and semimembranosus, which are to be divided close to their points of insertion into the fibula and tibia. In the foot, the tendo Achillis is most frequently divided, as are also occasionally the tendons of the anterior and posterior tibial and peroneal muscles, although it seems to me that this injures the subsequent mobility of the foot. In straightening anchyloses, tenotomy was formerly very often resorted to; but for this purpose it may be entirely dispensed with. In anchylosis of the knee-joint, for instance, if the above-named muscles be not united to a cicatrix, they may be gradually stretched during anæsthesia, that is, if they be still muscles and not strings of pure connective tissue, as is rarely the case. I shall not here speak of tenotomy of the ocular muscles, the operation of strabismus, as this is treated of in ophthalmology. Sometimes, also, we may be obliged to divide tendons in antagonistic contractions, for the purpose of rendering the contracted muscles inactive for a time, and subsequently elongating their tendons by extension, to give the paretic antagonist more play and less work; the latter are then opposed by no force, or, at least, by a weaker one so that equilibrium is restored. Of course, this is only to be done for muscles whose antagonists are not entirely paralyzed, but only paretic; in perfect paralysis, tenotomy of the contracted muscles would have no effect. The revivifying action of tenotomy is occasionally spoken of; it is to the above cases that this expression refers; indeed, in antagonistic contractions the action of tenotomy is sometimes astonishing.

The subcutaneous *division of fasciæ* is not much done; the cords of the fascia lata, which form when the thigh is kept flexed, are often divided with benefit, as it is difficult to stretch them; the fascia plantaris may also be occasionally divided with benefit, when it is tense, in club-foot. Division of the fascia fails in the cases where we might use it with most benefit, that is, in contraction of the palmar fascia. From *Dupuytren's* description of the results of this operation, in spite of the warning of my former preceptor, I was once led into performing it; but it was followed by such extensive suppuration that I was glad when this finally ceased. In spite of all orthopedic after-treatment, the hand finally remained as it had been; some slight improvement soon disappeared again, and I believe that this affection, in its higher grades at least, is incurable.

*Division of ligaments* is rare; but in club-foot I have often divided the small ligaments of the ankle-bones, if they were tense; and, in spite of the fact that I must certainly have frequently opened the small joints subcutaneously in so doing, I never saw any bad results. *B. von Langenbeck* introduced division of the external lateral ligament of the knee in genu valgum; in this the knee-joint is always temporarily opened. This operation is only proper in the highest grade of the affection, but greatly aids the treatment; I had not previously seen it, or even thought much about it, fearing that it might be followed by suppuration of the knee-joint; a few years since, in one case, I did the operation on both knees of a young man who had excessive genu valgum; the wound healed without any inflammation of the knee-joint, and the orthopedic treatment was very quickly concluded. The patient went out of the hospital with his legs perfectly straight. On the whole, the operation is rarely indicated. So far as I know, no other ligaments are divided.

It was natural to think of dividing contracting cicatrices also, so as to stretch the new cicatrix; but would it not be wiser not to let the cicatricial contraction come to such a point as to impair function? Would it not be best, even during the healing of a large wound—in the bend of the elbow, for instance—to keep the arm extended, so that it should not be contracted by the cicatrix? The idea is a good one; but the result rarely corresponds to such a tedious treatment, for, in the first place, such wounds, in which there can be no cicatricial contraction, heal with great difficulty, and, when they are finally healed and the limb is set free, contraction nevertheless occurs. I well remember a child with such a wound in the bend of the elbow, from a burn, which, as assistant in the Berlin clinic, I had to dress daily. The arm was kept extended on a splint, and took six months to heal; finally, the child was discharged, with the arm perfectly movable and the wound healed, and I was very proud of the cure. Two months later I saw the child, with the cicatrix entirely contracted; the arm was at an acute angle, and almost immovable. Subsequently I lost sight of the patient, and do not know what was the final result; but I clearly saw that I had worried myself and the child for months in vain. Several similar cases have radically cured me of the idea that we can, in such cases, do much by orthopedic treatment during the cicatrization of the wound. I advise you to let the wounds heal as they will; large wounds, from burns in children, will even thus give you enough trouble, as they always heal with difficulty, and readily assume an ulcerative character. In the course of months, often not for years, as its vessels are obliterated and its tissue becomes more like subcutaneous tissue, the cicatrix loses its rigidity, becomes more distensible, tougher, more elastic; hence, with time, mobility increases, in case it has been impaired by the cicatrix. You have already been told how you may aid this atrophy of the cicatrix by compression and distention. When the cicatrix has finally been reduced to the smallest size, you may occasionally, with advantage, excise the whole or part of it, at intervals, always being careful to obtain healing by the first intention, so that, in place of the thick, scarcely-distensible cicatricial string, you may have a fine linear cutaneous cicatrix, which may be stretched more readily than the old cicatrix; but if you have suppuration and gaping of the wound after these operations, the result is very doubtful (as, under the same circumstances, in tenotomy); there again forms a broad, granulating, slowly-healing wound, and a cicatrix as broad, long, and firm as the previous one. Hence you can only advantageously excise contracted, string-like, thin cicatrices. In removing complete, broad cicatrices, such as occur in the neck after burns, excision is not enough; a portion of distensible skin from the vicinity must be made to grow in the place of the cicatrix. This may be done by sliding a piece of neighboring skin, or by transplanting a flap of skin, according to the rules of plastic surgery, which I shall not enter into here.

We have now to speak of the treatment of distortions due to antagonistic muscular contractions; I have already told you that tenotomy may be useful in these cases also, but it is only an adjuvant to the treatment; the essential point is the removal of the paralysis. The curability of these contractions, and of the deformities they cause, will depend on what we can do for the paralysis. Here opens the wide field of neuropathology, with which you will become better acquainted in the lectures on medicine, and in the medical clinic. There are many cases where you would at the outset give up any treatment of the paralysis; in tumors of the brain, apoplexies, chronic encephalitis, traumatic injuries of the spinal medulla, extensive injuries of nerves, etc., treatment will do little good. Other cases of spinal disease with paresis of the lower limbs, especially in children, sometimes give a relatively good prognosis. On the one hand, treatment with cod-liver oil and iron, malt or salt baths, and especially time, may act very advantageously in removing the changes in the spinal medulla, of which we unfortunately know but little; on the other hand, irritations may be applied to the muscles themselves, that may revivify them; we may expect relief in those cases especially where there is no complete paralysis or paraplegia, but only paresis of certain groups of muscles. Here two external remedies are the most useful: 1. *Gymnastic treatment;* 2. *Electricity.* The former consists in awakening the slumbering, slightly-developed contractile power by concentrating the will on the paretic muscles. Certain movements are made regularly at certain times; this may be well done by the "Swedish movement-cure" that has been recently introduced: this consists in requiring the patient to make movements with certain muscles, while the gymnast offers a slight opposition. For instance, I hold your arm extended; you now bend it, while I oppose the movement by gentle pressure; of course, the proper movements must be determined for each individual case. Of late, this method of gymnastics has become quite popular, and proved useful; evidently it, like all gymnastics, is useless in complete paralysis.

Our second remedy is electricity; of late great advances have been made in its use. The apparatus employed has been greatly simplified, rendered more transportable, and so adjusted that the current can be strengthened or weakened at will. Moreover, the methods in which electricity is applied are greatly improved; formerly one or several groups of muscles of a limb were electrified, by applying the poles first on one place then another; now we understand electrifying the individual muscles; the French physician *Duchenne de Boulogne* has done great service in this matter. The points at which the pole or poles should be applied to induce contractions in the different muscles were first found empirically by *Duchenne;* subsequently *Remak* discovered that, as a rule, it was at the point where the largest motor nerve entered the muscle. Of late, *Ziemssen* has been most successful in electro-therapeutics; his book is characterized by practical utility and scientific importance, and above all by its trustworthiness. The treatment is so carried out that usually one or two sittings are had daily, during which first one, then another, muscle is methodically electrified; this may be continued half or three-quarters of an hour, but not too long, for fear of destroying the weak nervous activity by too great irritation. Much harm might be done by excessive electrization; a physician should always conduct the treatment, and give very positive directions about the duration of the sitting, and strength of the current. Usually we very soon see how much the muscles contract to the electrical irritation when they perhaps cannot be moved spontaneously; we should not give up even if we do not obtain any twitchings at the first sitting; occasionally these only appear after a time, when the electricity has had some effect.

Of late, *Barwell* has successfully employed a very ingenious method for removing contractions; he makes continued traction in the direction in which the muscles fail to act; for instance, in club-foot, a stout india-rubber band is fastened to the outer border of the foot, and the inner side of the tibia close below the knee; this acts continuously as an "artificial muscle." This seems to me rational, and it should be tried extensively. I have used this method in several cases, with very quick result; *Lücke* and *Volkmann* have also stated recently that they have attained good results by this treatment.

In pareses, movement of a few muscles occasionally suffices to enable the patient to walk, if a certain firmness which the muscles fail to supply is given to the limb by some sort of a splint. These splints are not always to be regarded as a last resort, but they may aid the treatment by enabling the patient to walk alone with the aid of sticks. But the movements of walking, made by the paretic muscles, have an excellent gymnastic effect; although artificially supported, the patient in this way uses his muscles, while, if he were continually lying or sitting, the muscles would remain entirely inactive, and atrophy more and more. Machines are also serviceable in keeping the legs extended and the feet at the proper angle, thus preventing contractions.

Gymnastics, electricity, artificial muscles, and splint apparatuses, combined with proper internal treatment, especially suitable water-cure, may do a great deal for these patients; and, although many of them are incurable, some are curable, and others may be greatly improved.

[The hypothesis of *siren* formation advanced (page 615) is supported by the formation of the seal, so many of the stages of development of the human embryo correspond to what we find persisting in some of the lower animals. *Dieffenbach* had noticed that all new-born children had a slight degree of club-foot, and, as a fact, the rotation of the limbs is not completed at birth, nor till during the first year of life, or even later, when the child begins to walk. At first the toes point inward, so that when learning to walk children often go on the toes. You could not readily place a six-months-old child in the typical position of the soldier, with the heels together, toes turned out, and legs touching all the way up. As the rotation does not affect the foot alone but the entire limb, the knee and hip of the newly-born child are not identical with those of adults, and this difference is even more marked in congenital club-foot.

An important factor in the development of scoliosis, in addition to those mentioned (page 618), is the position assumed in certain occupations, as in writing, etc. If the school-benches or tables be improperly constructed, the body may be kept daily for hours in an unnatural position. The longer the study-hours, the less time given to recreation and exercise, the greater the effect of this extra fatigue; so that finally the faulty position, which was at first assumed merely during the occupation, becomes habitual, and the spine does not resume its normal shape. At first by traction the curvature may be straightened out, or the children may do it voluntarily, but after a time this is not readily accomplished.

Quite recently *Mikulicz* made very thorough investigation of the statistics of genu valgum, which proved that the first changes showed in the diaphyses of the femur and tibia, and that the deformity was due partly to uneven growth at the epiphyses, partly to abnormal curvature of the diaphyseal ends. At first the knee-joint with all its parts are not implicated, so that the epiphyses appear set on to the abnormally curved ends of the diaphyses. Genu valgum is always accompanied by rachitis of the limbs. In high grades, besides the already mentioned anomalies of growth, there is irregular proliferation of the epiphyseal cartilages, the inner one increasing, the outer diminishing, and thus increasing the deformity. Through these influences combined, the height of the internal condyle of the femur is increased, while the external condyle does not grow; the internal lateral ligament elongates while the external contracts: as a consequence, the biceps muscle shortens and forms a tense cord.

Genu valgum is always accompanied by a pathological distortion of the foot; for, in spite of the angular position of the knee, to bring the sole of the foot to the ground the patient usually treads on the outer border of the foot, while the inner is elevated; so the metatarsus is twisted on its long axis, as in pes varus. When patients with genu valgus have been able by this manœuvre to correct the anomaly of the knee-joint, their gait is bearable and the inconvenience comparatively slight, even when the varus is consid erable; but the case is quite different in patients who have from the first walked on the inner border of the foot; they soon acquire marked pes planus, which renders walking nearly impossible. Severe pains occur in the foot, the gait becomes tottering and painful, and the combination of genu valgum and pes valgus not unfrequently induces the greatest functional disturbances of the lower extremity. Even slight grades of pes planus greatly impair the use of the leg.

Such cases are very frequent in young children, and may often be remedied by inserting a piece of beveled sole-leather in the shoe under the instep.

If *rigor mortis* has occurred while the legs were lying extended on a horizontal, level surface, the feet are always fixed in a position of slight equino-varus. The same is the case in persons who have lain long in bed without using their legs; if the legs be not moved, the elevation of the heel and rotation of the foot inward constantly increase.

After injuries or inflammation the tendons readily become necrosed, and as the dead tissue is only replaced by cicatricial substance, the tendon is shortened and the cicatrix is united with the sheath, which destroys the natural mobility of the tendon. Many such cicatrices in muscles are congenital, perhaps caused by the umbilical cord encircling a limb. Usually this amputates the limb, but at other times we find a cicatricial atrophy of the skin and disappearance of the muscle, which we must suppose to be replaced by connective tissue; this causes shortening of the muscle and distortions, particularly in the foot. Much more frequent are cases of caput obstipum from shortening of one sternocleidomastoid muscle from acute traumatic myositis; this inflammation is probably almost always caused by injury of the muscles by the forceps during instrumental delivery. In these cases immediately after birth, and during the first week of life, we may find an extensive, painful inflammatory swelling of the muscle, which disappears spontaneously, usually leaving cicatricial shortening, but sometimes going on to suppuration and formation of a true abscess of the muscle, which of course heals with a shrinking cicatrix.]

# CHAPTER XIX.

## *VARICES AND ANEURISMS.*

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### LECTURE XLIII.

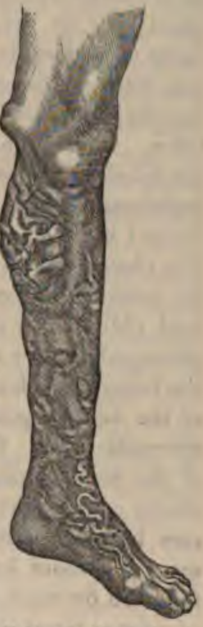
Varices: Various Forms, Causes, Various Localities where they occur.—Diagnosis.—Vein-stones, Varicose Lymphatic Vessels, Lymphorrhœa.—Treatment.—Aneurisms: Inflammation of Arteries.—Aneurysma Cirsoideum.—Atheroma.—Various Forms of Aneurism.—Their Subsequent Changes.—Symptoms, Results, Etiology, Diagnosis.—Treatment: Compression, Ligation, Injection of Liquor Ferri, Extirpation.

By varices we mean distentions of veins; these may have various forms, and usually affect both the diameter and length of the vessel. Elongation is only possible when the vessel bends laterally, and takes a tortuous course, as also occurs in inflammation of the smaller vessels. In some cases the elongation is less marked, and the diameter of the canal is not regular, but the vessel is distended in a spindle or sack-like shape at different points, especially where the valves are. Most frequently the large veins of the subcutaneous cellular tissue are thus affected; sometimes chiefly the deep muscular veins, often both are alike affected. But there are also varicosities in the smallest veins of the cutis, which are scarcely visible to the naked eye, these are often the only ones affected; this gives an even, light-blue nodular appearance to the skin. As a result of this distention of the veins, which occurs very gradually, more serum than usual escapes from the capillary vessels, as the lateral pressure in them is greatly increased by the distention of the walls of the veins, and the consequent insufficiency of the valves. The thinning of the walls of the vessels, and the transuded excess of nutrient material, may be gradually followed by escape of wandering cells, and their organization to new tissue; thus we have a serous, then cellular infiltration, and thickening of the tissue traversed by the varices; red blood-cells may also escape through the capillary walls (*Cohnheim*). We have already explained

(Lecture XXIX.) how, by a further advance of this process, the tissue is more and more changed, and chronic inflammation and ulceration induced. In this way are developed not only ulcerations but also some other forms of chronic cutaneous inflammations, especially a chronic eruption of vesicles, "eczema" of the leg.

Now we must take up the question, What is the cause of varices? It is probable that the cause is an obstruction to the return of the venous blood, a pressure, compression, or narrowing of the calibre of the vessel in some way. But the obstruction cannot be of sudden origin, for this usually causes œdema; the same is true of ligation of a large venous trunk and rapidly-appearing thromboses. The pressure must then affect the vein gradually. Still, even this is not enough; often a gradually-increasing pressure does not cause varicose veins, but free collateral modes of escape form, so that there is no effect, or only a slight, indurated œdema. There must be a coincident tendency to dilatation of the vessels, a certain laxity or distensibility of the walls of the veins.

FIG. 113.



Varices in the part supplied by the great saphena vein.

Anatomical examination of varicose veins shows that the walls are absolutely thickened by deposits of connective tissue between the muscle-cells, but the latter do not seem increased, and, as the calibre of the vessel is six or eight times the normal size, they must prove insufficient to urge the blood onward, the more so as the valves do not grow as the dilatation goes on, and consequently soon prove insufficient. Up to the present time we have had no detailed histological investigations about the formation of varices, and especially about the relation of this disease to aneurism. In many cases the disposition to varices may be regarded as individual, in others it is inherited; diseases of the vessels are not unfrequently hereditary, those of the arteries, as well as of the veins and of the capillaries, by whose morbid dilatation the so-called mother's marks are caused, whose transmission by inheritance is known even to the laity. Hence, we can only regard the cause of varices, which we are about to mention, as exciting causes acting on an existing predisposition. The disease is more frequent in women than in men; the chief cause is said to be repeated pregnancies: the uterus, gradually enlarging, presses on the common iliac veins, and later on the vena cava, and occasionally this even induces œdema of the feet. Often there are varices in all the parts supplied by the saphenous vein; again, in those supplied by the pudic, as in the labia majora. It is far more difficult to find the causes of the more rarely-occurring varices in man. Large collections of fæces may, by pressure on the abdominal veins, prove an exciting cause of varices, but this is rarely seen. In many men with varices you will find disproportionately long lower limbs, especially long below the knee; in some cases this may also favor congestions in the veins. Possibly, also, the collection of hard fat, or else shrinkage in the falciform process of the fascia lata, may cause congestion in the saphenous vein, as the latter sinks into the femoral at this point. So far as I know, there are no anatomical investigations on this point. The obstruction to the flow of blood need not always be directly in the territory of the dilated veins: for instance, gradual narrowing and final obliteration of the femoral vein, below the opening of the saphena, might very readily cause enormous distention of the branches of the latter by collateral circulation. Varices occur at some other parts of the body, especially at the lower part of the rectum and in the spermatic cord. Varices of the hæmorrhoidal veins in the lower part of the rectum cause *hæmorrhoids*, which, as is well known, occur chiefly among persons who lead a sedentary life. The disease is very rare in other parts of the body; it occasionally occurs in the head, usually without known cause, it may form after an injury, if this be followed by union of the walls of the arteries and veins and passage of arterial blood into the veins; this would be a varix aneurysmaticus, of which we spoke in the second chapter. In the pathological anatomical atlas of *Cruveilhier* you find given as a great rarity a picture of large varices of the abdominal veins; there is a similar preparation in the pathological museum at Vienna.

The *diagnosis* of varices is not difficult when the cutaneous veins are affected; those of the deep muscular veins can rarely be diagnosed with certainty; in the leg and thigh the whole course of the tortuous veins is so evident through the skin that they may be readily recognized, but in other cases we see only a few light-blue, fluctuating, compressible nodules; these chiefly correspond to the sac-like dilatations of the veins, and to the points where the valves are. Here we occasionally find hard, round bodies, phlebolites or *vein-stones;* on examination, these prove to be nodules in layers, at first consisting of fibrine; they may subsequently calcify entirely, so as to assume the appearance of small peas. In the great majority of cases, varices of the lower extremities cause no difficulty, except, perhaps, a feeling of tension and heaviness in the limbs after long standing or walking. But in other cases there are occasionally thrombi in single venous dila tations; inflammation of the wall of the vein and surrounding cellular tissue follows, and, although, under early treatment, the inflammation usually terminates in resolution, suppuration or abscess may eventually develop. The treatment is the same as has been already given for traumatic thrombus and phlebitis. Another danger that may arise from varix is its rupture, a very rare occurrence; if the patient be kept quiet, the bleeding may be readily checked by compression, and there is no danger if medical aid be at hand. A varicose ulcer, in the strict meaning, may form from such a ruptured varix, but this is rare, for the wound usually heals quickly. If the skin and subcutaneous tissue of the leg be greatly indurated, and if this induration has also affected the adventitia of the cutaneous veins, they lie immovable, and, in the firm, leathery, rigid skin, they feel like half canals or gutters. I call your attention to this, as otherwise in such cases, from the induration of the skin, you might entirely overlook the varices.

The *treatment* of varices is very unsatisfactory, as we know no way of removing the disposition to this disease of the veins. Nor can we usually control the causes of the pressure; so we may really conclude that varices are not curable, i. e., we have no remedy for restoring the morbidly-dilated veins to their normal size. For some cases we must say that, physiologically considered, the formation of varices is Nature's mode of equalizing abnormal pressure in the vessels, and that we may not try to remove the varices till we can get rid of their causes, for, if we removed one or more of these morbid strings, others would form in their place. For this reason I reject all operations which aim at removing one or more varicose nodules from the leg. If you bear in mind that any operation on the veins may prove dangerous to life by complication with thrombosis or embolism, you will agree with me in considering the operation for varices entirely uncalled for. Nevertheless, these operations are often done in France, and not unfrequently prove fatal; there are many methods of operation, about which we shall say a few words. The oldest method, which was practised by the Greeks, consists in exposing the varicose veins, and either cutting or tearing them out. Later, the hot iron was applied to induce coagulation of blood in the veins, which resulted in obliteration of the vessels. We may also inject liquor ferri sesquichlorati with a small syringe having a needle-shaped nozzle, as you know this quickly causes coagulation of the blood. After this came the ligature of the veins, especially the subcutaneous ligature after *Ricord*, and the subcutaneous rolling-up, the *enroulement* of *Vidal*, little operations that I shall show you in the course on operations; these are very ingenious methods, but I am sorry to say they do not succeed, and are not free from danger.

But shall we do nothing for varices? Yes, we should try to keep them within certain bounds, and thus prevent or reduce to a minimum their bad effects. For this purpose there is only one remedy, *continued compression*, which, however, must only be used in such a degree as is bearable to the patient. We use two different mechanical modes of compression in these cases, the laced stocking and regular bandaging. The laced stocking consists either of a carefully-made, close-fitting leather stocking, split at one side, and laced up, like corsets, till it is tight enough, or else of a tissue of rubber thread, spun over with silk or cotton, of the same stuff that most suspenders are made of. These laced stockings, which must be very carefully made, and worn continually, are unfortunately quite expensive, and, as they cannot be washed, must often be replaced, so that they are only practically useful for persons of means. In most cases a carefully-applied roller-bandage suffices. For this purpose, you may best take a cotton bandage two or three fingers' breadths wide, soaked in good bookbinder's paste, and, excepting the heel, bandage the whole foot and leg; with care, such a bandage may be worn five or six weeks, and even if the skin be considerably infiltrated, it may prevent the formation of ulcers by obstructing the further development of varices.

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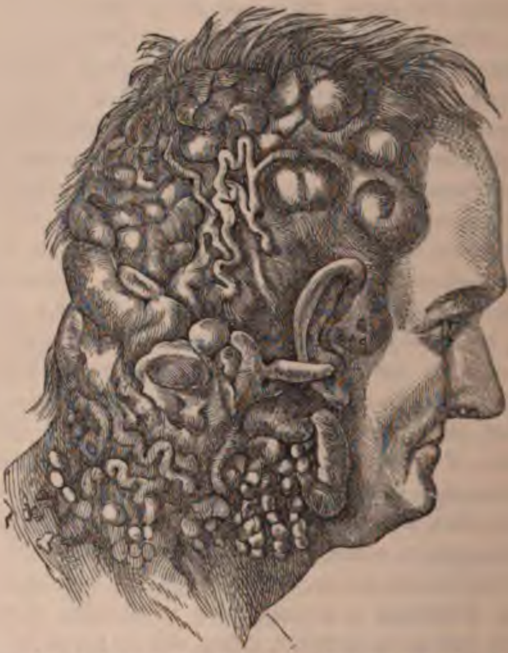
It is some time since we spoke of traumatic *aneurism*, but you will remember that we mentioned it under punctured wounds (page 143), and that I then told you an aneurism was a cavity, a sac, which directly or indirectly communicated with an artery; you already know that such sacs may develop from injuries of the artery by puncture, subcutaneous rupture, or contusion. But now we do not mean to speak of these traumatic, so-called false aneurisms, but of *aneurysma verum*, which develops gradually from disease of the wall of the artery. To explain to you clearly how this occurs, it will be best to start from the anatomical conditions. At present, you know but little of the diseases of arteries; the only ones that have been mentioned so far are thrombosis after injury, the development of collateral circulation, and atheroma, which we hastily spoke of when treating of senile gangrene. And these comprise almost the whole list, only that so far we have taken merely a one-sided view of atheromatous disease. Of the different parts of arteries the tunica muscularis and intima are most frequently diseased, and they seem to be affected primarily. The tunica media is composed of muscle-cells and some connective tissue; the tunica intima consists of non-vascular, elastic lamellæ, fenestrated membranes, and very thin endothelium. It may be readily shown that, after injury of an artery, its walls swell, and remain thickened for a time; the plastic infiltration of the walls may lead to suppuration, and small foci of matter may form in them, though this is seen more rarely in arteries than in veins. With these processes there is a relaxation of the membranes, the intima may be detached from the media more readily than usual, the latter is softened, the muscle-cells may in part disintegrate, and, as a result of this diminished resistance, there may be a dilatation of the artery. Such acute inflammations with plastic new formations and partial softening may doubtless occur spontaneously, and, although we have no special observations on this point, still, from analogy with other tissues, there is no doubt that a spontaneous, idiopathic, acute, and subacute inflammation of the arteries may run its course in this way, and probably occurs with acute inflammations of other tissues. At all events, these acute spontaneous inflammations of arteries are very rare; the chronic forms are far more frequent. One form of aneurism alone possibly depends on a more subacute inflammation of the artery, with diminished resistance of its walls; this is *aneurysma cirsoideum*, or *aneurysma per anastomosin*, also called *aneurysma racemosum*. This form of arterial dilatation is totally distinct from the aneurisms to be hereafter mentioned; in them there is not circumscribed dilatation of one part of an artery, but dilatation of a large number of arteries lying close together, which are, moreover, very tortuous, a sign that they have also increased in length. *Cirsoid aneurism is, then, a convolution of dilated and elongated arteries.* For these changes to occur, there must be a considerable new formation in the wall of the artery, longitudinally, as well as in the circumference; the dilatation is possibly due to atrophy of the muscular coat; usually (without, however, being able to prove it) paralysis of the walls of the arteries is assumed to be the exciting cause of this variety of aneurism; still, although paralysis might explain a moderate dilatation of the artery, we have nothing to explain the paralysis, and this would not render any more comprehensible the elongation of the artery, which can only depend on a new formation of the elements of the wall. As already stated, I think that this variety of arterial dilatation, which closely resembles inflammatory dilatation and looping of vessels, must be referred to an inflammatory change in the artery, and not to chronic inflammation with atheroma, to be hereafter described, but to a more subacute, diffuse inflammation. This view is supported by various etiological factors; these aneurisms not unfrequently develop after blows or other injuries; they are most frequent at points where numerous small arteries anastomose, as in the scalp, over the occiput, vertex, and temples; this variety of aneurism might be regarded as an excessively-developed collateral circulation; the collateral arteries, besides dilating, become tortuous; the process is evidently the same in both cases. We have also to mention that these aneurisms are particularly apt to develop in young persons, in whom the chronic diseases leading to other aneurisms are rare. The diagnosis of cirsoid aneurism is very simple, if, as is usually the case, it lies just under the skin; it has been found more deeply seated, as in the gluteal artery, but it is more frequent on the head; here we may feel, and occasionally see, the tortuous pulsating artery, so that the disease is readily recognized; it is not frequent.

FIG. 114.



Cirsoid aneurism of the scalp in an old woman; a small tumor was said to have existed at birth, and to have developed gradually to this size. After *Breschet*.

We have still to mention that the arterial wall may become diseased by a suppuration or ulceration extending from the neighboring parts, first to the adventitia, then to the other coats; this is the case more rarely in acute abscesses than in chronic ulcerations. As an example of this we see that, in the development of cavities in the lungs, it not unfrequently happens that the ulceration attacks the walls of the smaller arteries, and the adventitia is partly destroyed and softened. The result of this is, that the artery dilates at this point, and a small aneurism is formed, whose rupture causes severe hæmorrhage. Other ulcerations also may (though this rarely happens) find their way to an artery and destroy its walls, so as to induce bursting of the artery, and fatal hæmorrhage if the artery be a large one. I have seen several such cases: an old man had an abscess deep in the neck which opened into the pharynx; this was diagnosed from the gradual formation of a painful swelling in the neck and the free expectoration of badly-smelling pus; the patient had only been in the hospital a few hours when he threw up a large amount of blood, was quickly asphyxiated, and died; autopsy showed that, as a result of circumscribed suppuration of the superior thyroid artery, it had thrown out a quantity of blood which had passed directly into the larynx and caused suffocation. In another case in a young man who had caries of the right temporal bone, there were repeated hæmorrhages from the right ear; I diagnosed an abscess on the under side of the temporal bone with suppuration of the internal carotid artery. The bleeding could not be checked by tampons to the ear; I ligated the right common carotid. The bleeding ceased for ten days, then began again; after repeated tamponading and digital compression of the left carotid without permanent result, I also ligated the left common carotid; but in two days the patient died of profuse hæmorrhage from the right ear, nose, and mouth; the abscess, which was filled with blood, and could now be regarded as an aneurysma spurium, had also opened into the pharynx. The *post mortem* fully confirmed the diagnosis.

We now come to chronic diseases of the arteries and their results, to *true aneurisms.* In advanced age it is very common for the arteries to become exceedingly thick and hard and occasionally even looped, especially those of the diameter of the radial or smaller. If we examine these arteries more accurately, we find the tunica intima thickened, of cartilaginous firmness, it is more rigid than usual, and gapes; in places it is even as hard as chalk, or even quite calcified or ossified. The chalky parts are not diffusely spread through the walls of the artery, but form circles corresponding to the transverse muscles of the tunica media; it is the muscles of the vessels that ossify. In such persons, on the inner surface of the aorta and its first large branches, we find whitish-yellow spots, striæ or plates of chalky firmness, or rough as if gnawed, with their edges hollowed out. If we cut into these spots, we find the whole intima of cartilaginous hardness, whitish yellow, and completely calcareous or hard as bone, or else friable, granular, or pulpy. Where this disease has attained a high grade, the arteries become bulged out. This is atheroma of the artery as it appears in the cadaver. We often find the recent and old stage near together or in different arteries. If we examine these spots more carefully with the microscope, especially in fine cross sections through spots of different appearance, we find that the first changes occur in the outer layers of the intima, on the borders of the media; here a moderate grouping of cells begins. The young cells may lead to connective tissue and new formation and callous thickening of the arterial wall; but they are usually short-lived; while new ones appear in the periphery of the affected spot, the first ones disintegrate to a granular detritus, to a pulp formed of fine molecules and fat, which remains rather dry, as in caseous degeneration; the destruction thus slowly extends laterally, the nutrition of the media, as well as of the inner layers of the intima, suffers; the muscle-cells of the former become granular and fatty, as do the elastic lamellæ of the intima; the change thus progresses inward till the last lamellæ and the epithelial membrane are perforated, and the cavity filled with atheromatous pulp opens into the calibre of the artery. The atheromatous process, beginning as a hollow ulcer, has led to an open ulcer with undermined edges; you see the mechanism is the same that you have already seen in the skin and lymphatic glands; there is a chronic inflammation ending in caseous degeneration, or, as the pulp is called in this position, in atheroma. This is the essential part of the process, as far as concerns the development of aneurism; but there are some variations, from the different structures of the arteries. The less developed the muscularis and intima, the less atheromatous pulp will be formed, as this results chiefly from breaking down of the intima. To commence with the small arteries, whose diseases we may study in the microscopic cerebral arteries: here we find the collections of cells mostly in the adventitia, which is but little and only secondarily affected in large arteries. Almost the whole adventitia changes to cells, the few muscular cells atrophy, the fine hyaline membrane, which acts as intima, is very elastic; thus the softening of the adventitia, caused by the cell-infiltration, soon induces dilatation and finally bursting of the artery, as the walls are no longer sufficiently firm to resist the pressure of the blood. Occasionally also there is a plastic production of adventitia; club-shaped vegetations form, which consist partly of newly-formed fibrous, partly mucous connective tissue. We cannot here discuss this further, especially as it does not affect surgery. A fatty degeneration and calcification of the muscular coat also occur along with the plastic infiltration of the adventitia in the smaller cerebral arteries, but are not frequent. Let us pass to arteries the size of the basilar, radial, etc. Here the plastic process in the adventitia occasionally still combines with those in the other coats, although pulpy disintegration and calcification of the latter do occur. Sometimes there are thickening and looping of these arteries, sometimes disintegration and softening, with consequent dilatation or aneurism; for, when the media and adventitia become softened to atheroma pulp at some point, the adventitia is no longer strong enough to resist the pressure of the blood, and it bulges. If we now turn to the large arteries, aorta, carotid, subclavian, iliac, and femoral, in which, you know, the muscular coat is reduced to a minimum, or is even occasionally wanting, while the intima is composed of a large number of elastic lamellæ, and lies almost immediately on the adventitia, which has more or less elastic filaments—here there is least plastic process in the adventitia; the pathological change, the disturbance of nutrition, evinces itself chiefly in rapid breaking down or calcification of the pathological new formation, which occurs partly on the borders of the intima, partly in that coat. As already mentioned, cases do occur where extensive circumscribed connective-tissue new formations occur in the intima in the form of cartilaginous callosities; but this is rarer than the change to atheroma-pulp. In the last mentioned arteries true atheroma pulp forms most frequently, hence aneurisms are most frequent in them. If you examine this atheroma-pulp microscopically, besides the above-mentioned molecular and fat granules, you find fat-crystals, especially of cholesterine, and crumbs of carbonate of lime, also hæmatoidin-crystals, which come from blood-clots depositing on the roughnesses in the arteries, but the hæmatoidin develops from their coloring matter.

You have now a general view and description of atheroma in arteries of various calibre, and can now understand how, by softening the walls of the vessels, it may lead to partial dilatation of the artery, or aneurism. The form of this dilatation may vary somewhat, according as the whole periphery of the artery is regularly diseased or not, and as softening or calcification predominates.

The dilatation of the artery may for some distance be perfectly regular; this is called *aneurysma cylindriforme ;* if the aneurism be more spindle-shaped, it is termed *aneurysma fusiforme.* If the softening be limited to one side of the arterial wall, we have a sac-like dilatation, *aneurysma saccatum*, which may communicate with the calibre of the artery by a larger or smaller opening. A further variety in the formation of the aneurism may arise from all the coats regularly participating in the formation of the aneurism, or from the intima and media being entirely softened and destroyed, so that only the gradually-thickening adventitia and infiltrated surrounding parts form the sac. Finally, under the last conditions the blood may press in between the media and adventitia, separate the two coats, as if the layers of the artery had been dissected up anatomically; this is called *aneurysma dissecans.* These divisions may be carried still further, but practically they have very little value. I shall only mention in addition that, on subcutaneous bursting of an aneurism composed of all the arterial coats, it assumes more the anatomical peculiarities of an aneurysma traumaticum or spurium. A short time since I saw an apparently healthy man, about fifty years old, who, when turning in bed, had an enormous tumor develop in the thigh, which soon proved to be a diffuse traumatic aneurism; I had no doubt that the femoral artery was diseased, and had suddenly burst at some point in the middle of the thigh. After compression had long been used in vain, the femoral artery was ligated; it proved to be covered with yellow spots; the ligature healed well and became detached in four weeks, still the aneurism became larger and painful; the sixth week after the ligation gangrene of the foot began; I then made a high amputation of the thigh; the patient recovered. There was an immense aneurysma spurium, and an opening an inch long in the atheromatous femoral artery, which was not aneurismatic.

The further fate of the aneurism, and its effect on neighboring tissues or the extremity affected, are very important. As regards the anatomical changes in and about an aneurism, one is its increase in size, which not only displaces the neighboring tissues, but, by its pressure and pulsation, causes them to atrophy; this refers not only to the soft parts but to the bones, which are gradually broken through by the aneurism; the last effect is especially apt to be induced by aneurisms of the aorta and anonyma, which may induce atrophy of the vertebræ, sternum, or ribs. A further accompaniment is inflammation in the immediate vicinity, which, however, rarely leads to suppuration, often becomes chronic, and very seldom induces gangrene of the anurism. Lastly, there is often coagulation of blood in the aneurism; hard layers of fibrine may form on the inner surface of the sac, and at last entirely fill it, and so cause a spontaneous obliteration, one variety of cure of the aneurism. The worst accident is when the aneurism increases in size, and finally bursts; this may take place outwardly, but more frequently, especially in the large arteries of the trunk, it is inward, perhaps into the œsophagus, trachea, thoracic or abdominal cavity; sudden death from hæmorrhage is the natural result.

It is not our present object to show what may be the results of aneurism of arteries of internal organs; I shall merely mention that particles may be detached from the clots which form in the aneurismal dilatations, or on the roughnesses of the atheromatous arteries, and may pass as emboli into the peripheral arteries. These emboli occasionally cause gangrene; but this is not so frequent as is believed, for usually the coagulæ in aneurisms are firmly attached.

We shall now investigate more carefully *aneurisms of the extremities.* At first, they cause slight muscular fatigue and weakness, more rarely pain in the affected limb; if there be inflammation about the sac, of course there are pain, redness of the skin, œdema, and disturbance of function, which may go so far as to render the limb entirely useless if the aneurism continue to grow, and there be continued chronic or subacute inflammation around it. The formation of extensive coagulæ in the aneurism of a large artery may be followed by gangrene of the whole limb below it.

When speaking of gangrene, it was mentioned that it might result from atheroma of the artery, as so-called gangrena spontanea; but there the case was somewhat different: the small arteries were diseased; these lose their power, from destruction of their strong muscular coat, and can no longer urge on the blood, as they cannot contract. But here there is obliteration of an arterial trunk by coagulæ from an aneurism. I will relate to you a case observed in the Zürich surgical clinic. A man twenty-two years old, emaciated and miserable, was brought into the hospital; his right leg, nearly as high as the knee, was bluish black, the epidermis peeled off in shreds; gangrene was unmistakable. Examination of the arteries showed a spindle-shaped, pulsating aneurism of the left [right?] femoral artery, close below Poupart's ligament; there was a second one, three inches below, on the same artery; this felt hard; there was a third one in the bend of the knee, just as hard, but, from the swelling of the surrounding parts, the form could not be exactly made out; between the second and third aneurisms the artery continued to pulsate the first day the patient was in the hospital; the line of demarcation was not formed, it appeared likely to extend higher; gradually the pulsation ceased as high as Poupart's ligament; the patient died about a fortnight after his admission to the hospital. The autopsy showed the aneurisms that had been recognized during life, and also extensive atheroma of almost all the arteries. Taking this with what I told you, when speaking of the ligation of large arteries, about the development of collateral circulation, you will think there is a contradiction. Why does not gangrene occur when you close an artery by a ligature, as well as when it is blocked by a clot? The answer to this is, that a free collateral circulation sufficient for the nourishment of the peripheral parts only takes place when the arteries are healthy and capable of distention. But, when a coagulum passes from an aneurism into the artery, the neighboring arteries are usually diseased and not distensible, being calcified, or already partly obstructed; moreover, the closure of the artery is not, as in ligation, limited to a small space, but is very extensive, perhaps even, as in the case above mentioned, involving the whole artery; then there is no possibility of a collateral circulation, either by the direct route or by neighboring branches. The arteries must be very generally diseased, and the coagulation very extensive, to cause gangrene, so that it is not very frequent in aneurism; that would also be very unfortunate for the treatment, which, however, as you will hereafter see, chiefly has for its object the obliteration of the aneurism, with or without ligation of the artery.

We now come to the *etiology* of aneurism. Although atheroma is a very frequent disease of old age, and occurs everywhere, aneurism is by no means confined to old persons. In Zürich, atheroma of the arteries in old persons, and gangrena senilis, are quite frequent, but aneurism of the extremities is rare. The occurrence of aneurism is curiously spread over Europe: in Germany, aneurism of the extremities is rare; it is somewhat more frequent in France and Italy, and most frequent in England. It is difficult to explain this, only it is certain that diseases of the arteries, in common with rheumatism and gout, are more frequent in England than in any other country of Europe. [During the past five years (1865–1870), of 11,344 cases of disease and injury, in the New-York Hospital, there were 33 cases of aneurism, or about one case to every 344 patients. Of these there were: of the thoracic aorta, 6; abdominal aorta, 10; innominate artery, 1; subclavian, 2; iliac, 1; femoral, 4; popliteal, 8; not named, 1.] As regards age (of course we are not speaking of traumatic aneurisms), the disease is rare before the thirtieth year, more frequent between thirty and forty years, and most frequent after the fortieth year; men are more disposed to aneurisms than women. Special causes are little known; popliteal aneurism is most frequent among those in the extremities; the explanation of this has been sought in the superficial position of the popliteal artery, in the tension to which it is subjected on sudden movements, contusions, etc.; thus this form is said to occur especially often in England in footmen who stand behind the carriages; but I must acknowledge that to me this seems as improbable as the explanation given for chamber-maid's knee. I am inclined to believe that the tendency to diseases of the artery, as to gout, is due to hereditary influence; hard work and free use of liquor are also given as causes; in England especially, the latter is said to induce relaxation of the walls of the artery, even without atheroma.

The *diagnosis* of an aneurism of the extremities is not difficult, if the examination be careful and the aneurism not too small. There is an elastic, more or less hard, circumscribed (except in false or ruptured aneurism, which are diffuse) tumor connected with the artery; the tumor pulsates perceptibly to the sight and touch; on applying the stethoscope, you may hear a pulsating murmur, caused by the friction of the blood on the coagulum, or in the opening of the sac, or by the ricochetting of the blood in the sac. The tumor ceases to pulsate if you compress the artery above it. These symptoms are so striking that it might be thought the diagnosis could not be mistaken; still, errors have been made even by experienced surgeons, at times when they did not think of the possibility of aneurism, and were hasty. For, when the surrounding parts are much inflamed, the aneurism may be greatly masked by the swelling; it may be taken for a simple inflammatory swelling or abscess; it may even have originated from an abscess, as before stated. The latter mistake is the most frequent; it is punctured, and—what a disagreeable surprise—instead of pus, we have a stream of arterial blood. There is nothing at hand to arrest the hæmorrhage; the situation is shocking, even if the cool surgeon have presence of mind enough to make instantaneous compression till he decides what next to do. But I will not picture affairs too dismally; and I repeat that, on careful examination, such an error would scarcely be possible. If the aneurism be distended with clots, the pulsation of the tumor may cease, or be very indistinct, as may also the murmur; even here, however, further accurate observation will lead to a correct judgment. On the other hand, a tumor of a different sort may be mistaken for an aneurism. In the bones particularly, there is a sort of soft tumor (central osteosarcoma) which is very rich in arteries, and consequently pulsates distinctly. Numerous small aneurisms may form on these arteries, from the softening of the substance of the tumor and of the walls of the arteries; the sum of the murmurs in these small aneurisms may resemble a typical aneurismal murmur; in these cases also, only the most accurate examination and observation can show us the true state of the case. These pulsating bone-tumors are often regarded as true aneurism in bone. I do not believe there is any spontaneous aneurism in bone, but consider all these so-called bone-aneurisms as soft sarcoma in the bone very rich in arteries. Lastly, we may be tempted to regard a tumor, lying very near an artery and moved with the arterial pulse, as an independently-pulsating tumor, or an aneurism; the absence of the aneurismal murmur, the consistence of the tumor, the possibility of isolating it from the artery, and the further observation of the course, will guard you from error. The *prognosis* of aneurism varies greatly with its locality, so that nothing general can be said of it.

We now turn to the *treatment*, remarking, first, that in rare cases aneurism may recover spontaneously, by complete closure of the sac and a part of the artery by coagula; the tumor then ceases growing, and may gradually subside. As before mentioned, also, inflammation around the tumor may lead to local gangrene; if the artery has previously been occluded, the whole aneurism may become gangrenous, and be thrown off without hæmorrhage. These natural cures are very rare, but indicate the mode of treatment. I shall not here speak of the medical treatment of aneurism, except to mention one method, *Valsalva's*. The aim of this is, to reduce the volume of blood in the body to a minimum, so as to weaken the heart's action, and favor the formation of coagula. Repeated venesections, purgatives, absolute quiet, low diet, digitalis internally, and ice locally over the tumor, are the remedies with which the patient is treated under this method; the results are doubtful: the patients are very much debilitated, and the symptoms may then be less; but, as the patients regain their strength, the former condition generally returns. We may employ the above remedies to a moderate extent in alleviating severe symptoms in internal aneurisms, but they will not induce an actual cure; unfortunately, internal aneurisms must almost always be regarded as incurable. Let us pass to the surgical treatment of external aneurisms. This may be conducted in two ways; it may aim at the destruction of the aneurism, or its complete removal. In most cases the destruction of the tumor will be enough. The remedies for this purpose vary.

1. *Compression.* This may be applied in various ways: *a*, on the aneurism; *b*, on the affected artery, above the tumor. The latter is by far the most effective method, because even a moderate pressure on the aneurism is often painful, and may cause inflammation in its vicinity. The mode of employing compression also varies; it may be continued, and complete or incomplete; it may be temporary, but complete, i. e., such as to arrest the pulsation. The methods of compression are about as follows: *a*, compression with the fingers, particularly recommended by *Vanzetti*, and used by other surgeons with advantage; it is made by the surgeon, nurses, or by the patient himself, at intervals, so as to arrest pulsation completely for a few hours; if the patient can bear it, this is continued for days, weeks, or even months, till the aneurism no longer pulsates, and has become quite hard; *b*, compression of the aneurism by forced flexion of the extremity; this procedure, first used by *Malgaigne*, is particularly suited for popliteal aneurism; the limb is fastened in the position of extreme flexion by a bandage and retained thus till the pulsation in the aneurism has ceased *c*, compression with special apparatus, pads, compresses, etc., which must be so made that the pressure may be as much as possible on the artery, and that œdema may not be induced by simultaneous pressure on the vein; the pressure need not be hard enough to arrest pulsation entirely, but merely to diminish the supply of blood. Views regarding the efficacy of compression in the treatment of aneurism vary. Irish surgeons laud it highly; French and Italian surgeons

incline to it more than formerly; especially since the investigations of *Broca*, intermittent digital compression has shown some brilliant results. I think that, in most cases of aneurism, compression should be first resorted to; but observation shows that it is not alike suited for all cases, and is not of radical benefit in all. [Mr. *Coote* reports a case where aneurism of the innominate artery was cured by the application of a bladder of ice.]

2. *Ligation* of the artery. This may be done in various ways: *a*, close above the aneurism (after *Anel*); *b*, far above the aneurism, at a point of election (*J. Hunter*); *c*, close below the aneurism, i. e., at its peripheral end (after *Wardrop* and *Brasdor*). Of all these methods, ligation close above the aneurism is proportionately the most certain; ligation close below it the least certain. Ligation at a distance from the aneurism will cure the disease for a short time, occasionally even permanently, i. e., the pulsation in the aneurism will cease, but, when the collateral circulation develops fully, the pulsation may begin again. I have myself seen such a case; from a puncture with a penknife, a boy twelve years old had an aneurism the size of a large walnut in the femoral artery, about the middle of the thigh; the femoral was ligated close below Poupart's ligament; in ten days the ligature cut through, and there was great hæmorrhage, which, however, was instantly checked; then, after dividing Poupart's ligament, a second ligature was applied half an inch higher; this ligature held well; the wound healed; when the patient left the hospital there was again pulsation in the aneurism, which had previously become perfectly hard, and had ceased pulsating. But, in spite of such relapses, ligation remote from the aneurism will retain its importance, and continue the chief method, for, in the vicinity of the aneurism, the artery is occasionally so diseased that it is not advisable to ligate there; for the rigid and ossified artery might be so quickly cut through by the ligature that the thrombus would not be firm enough when the ligature falls.

3. Remedies which are supposed to directly induce coagulation of the blood in aneurisms. Of these, injection of liquor ferri sesquichlorati, after *Pravaz* and *Petrequin*, is relatively most frequently used; it must be done very carefully: it should be made with a small syringe, whose piston is moved by a screw, with every turn of which a drop escapes; a few drops of the liquor ferri should thus be very carefully forced into the tumor. Simple coagulation and shrinking of the aneurism may, and it is said do, follow this; but experience has shown that it is more frequently followed by inflammation, suppuration, and gangrene. I think that the action of the injected liquor

ferri is misunderstood; for it is not probable that a clot made by this substance becomes organized; it most likely merely irritates the wall of the vessel, causing it to inflame, and thereby lose the power of keeping the blood fluid (*Brücke*), thus secondarily inducing coagulation of that fluid and atrophy of the walls of the artery. *Von Langenbeck* injected a solution of ergotin into the immediate vicinity of an aneurism and cured it. I explain the action here also as being an inflammation of the wall of the vessel, with the results above mentioned. Electropuncture, nearly abandoned for a time, has been again resorted to by *Ciniselli*, and with very good results, even in aortic aneurism; a needle is to be passed into the aneurism and connected to the negative pole of a galvanic battery, while the positive pole is to be applied to any part of the body. [In an interesting case reported by Dr. *R. P. Lincoln*, in the *Medical Record*, the current was passed *directly* through needles introduced into the tumor.] Formerly it was thought that the galvanic current had the power of coagulating the blood directly. [This would seem to have occurred in *Lincoln's* case, as clotted blood escaped from the needle-punctures.] Physiologists do not consider this the mode of action, but think that the thermic effect of the current causes a small eschar around the needle in the aneurism, and that the clot forms around this. If we pass several fine needles into an aneurism and leave them twenty-four to forty eight hours, they also will cause inflammation and formation of a clot. [At a meeting of the New York Pathological Society, Dr. *Gurdon Buck* presented a specimen where needles had been used, and spoke of one where silk sutures had been employed; he gave it as his opinion that, in view of the liability to inflammation, such procedures were inadmissible in arteries near the heart.]

4. We now come to the mode of operative treatment of an aneurism which aims at its *complete destruction;* if this succeed, it is of course, more certainly a radical cure than the modes above described, but it is a much more serious operation. It may be done, according to *Antyllus*, as follows: The artery is to be compressed above the aneurism, then the whole sac is slit up and the coagulum turned out; through the sac probes are passed into the upper and lower ends of the artery, which is then ligated, the probes of course being removed—they are only intended to facilitate finding the artery; this operation, which I have seen performed several times for aneurisms resulting from venesection, is not always as simple as it appears, for it is not at all times easy to find the openings of the artery in the sac filled with coagulum, and often other arteries besides the main one bleed, as a collateral circulation occasionally opens into the aneurism.

After the operation there is suppuration of the whole aneurismal sac; in three cases of traumatic aneurism of the brachial, and one of the radial artery, I saw healing occur without any accident. If the aneurism be small and distinctly bounded, we might first ligate above and below, then extirpate the aneurism as we would a tumor. Of late, *Syme* has employed the method of *Antyllus* successfully in large arteries also.

I should like to give you some definite advice about the choice of method among these different plans of operating, but this is scarcely possible, as one plan or another will best suit different cases. In general terms, I can merely repeat that of late so many favorable results from compression have again been published from different sources, that it should not be too quickly abandoned. If, however, as usually happens in aneurisms from venesection, there be great diffuse swelling of the entire arm, the method of *Antyllus* appears to me preferable to all others; with good assistants it is very practicable, and is not so dangerous as is claimed by many persons. When we do not wish to make *Antyllus's* operation, we may try *Anel's* or *Hunter's*. I have least to say for the injection of liquor ferri in ordinary cases of spontaneous and traumatic aneurism. In varicose aneurism and aneurismal varix, ligating the artery above and below the opening will be the most certain method.

[In the diagnosis of external aneurisms we may place the hand, with the fingers slightly spread, over the tumor; if the pulsation is not aneurismal the hand is simply moved, but if it be so the fingers are separated still more. In the former case the pulsation is due to a cavity filled with fluid, or some other tumor pressing on an artery.

For treating aneurisms by compression one of the simplest plans is by means of a rod with its upper end against the ceiling or a framework over the bed, and the lower end on a cushion lying on the artery; the vessel is thus compressed by the rod till the pulsation ceases.

Another method is compression of the limb till it is bloodless, by *Esmarch's* elastic bandage. This may be applied daily with moderate pressure from the periphery to a point above the aneurism; then apply an elastic tube and remove the bandage; thus the part supplied by the diseased artery is cut off from the circulation. Complete compression is maintained from three to five minutes at first, and later for a longer time. Before removing the tube the elastic bandage should again be applied so as to make slight pressure, and then the blood be allowed to enter the artery. Thus complete compression is aided by moderate pressure in the intervals.

When an operation has been performed under *Esmarch's* bandage, after ligating all the vessels we can find, we may tampon the sac with carbolized gauze, apply an exact compressive bandage from the periphery upward, and then remove the tube. Then elevate the limb to facilitate venous circulation. Sometimes there is suppuration from the sac, particularly if all clots have not been removed; but this does not hinder cicatrization.

In ligating atheromatous arteries it is advantageous to use ligatures of material which may heal in the tissue, because this enables us to unite the incision by first intention. Instead of catgut ligatures, which are uncertain, as they sometimes absorb quickly, I use fine silk ligatures that have been soaked an hour in a four-per-cent solution of carbolic acid and then kept in antiseptic fluid in a well-covered vessel. This silk causes as little reaction as catgut, and gives all possible security against rapid solution of the ligature before the thrombus is organized.]

We must still add a few remarks about the treatment of cirsoid aneurism. The above methods of operation are only partially applicable to it. Direct compression of the entire tumor may be made by means of bandages and compresses prepared for the special cases; we mean particularly the aneurisms of this variety coming on the head which are the most frequent, but compression has rarely proved successful. The injection of liquor ferri may here prove useful, for suppuration or gangrene of the entire convolution of arteries is not so much to be feared as in aneurisms of the large arteries of the extremities. The destruction might be accomplished by ligating all the afferent arteries, but this is very tedious and uncertain; the result would be just as doubtful, and it might be dangerous to ligate one or both external carotids in a cirsoid aneurism of the scalp. Another method, having the same object, is to insert insect-needles through the skin at different points around the tumor, and apply a thread, as in the twisted suture; the result will be suppuration and obliteration, perhaps partial gangrene of the skin. Total extirpation may occasionally be resorted to; it is done as follows: Around the tumor we make numerous percutaneous mediate ligations close together; then we may cut out the main body of the tumor, with the dilated arteries, without hæmorrhage; this is the most certain and radical operation, but cannot well be resorted to when the tumors are very extensive; then we might try mediate ligation for different parts, and attain our end by partial extirpations. After his very thorough investigations about the treatment of these aneurisms, *Heine* also speaks very decidedly in favor of their extirpation.

Careful examinations of veins by *Soboroff* have shown that their walls are in very different conditions; he examined especially the saphenous vein and its branches (Fig. 113); he found that normally in different persons its layers varied essentially, and that even adjacent parts of the same vein were not exactly alike. This is very interesting, for it explains why the occurrence of varices is so unequally induced by the same cause, and is due to purely individual circumstances. Among varicose veins we may distinguish those with thin and those with thick walls. The enlargement of the muscular filaments and the lack of change in the endothelium are common to all. The variation in the diameter of the walls of the veins is chiefly due to thickening of the adventitia, whose vessels also increase, and of the cement which unites the muscular filaments; slightly also to thickening of the intima; but sclerosis of the latter, as in arterial sclerosis, is very rare. Hence, under increased pressure the anatomical conditions in the walls of veins are the same as in the urinary bladder and heart under similar circumstances. At first, in consequence of increased functional demands, the muscular filaments seem to grow; if then nutrition is increased by increase of the vasa vasorum, the connective tissue, especially the adventitia, is decidedly increased; if the nutrition be not increased, there is atrophy and total relaxation.

[At present, with the aid of antiseptic treatment, operations for varicose veins, after being for a time partly abandoned, have again been resumed. After applying *Esmarch's* bandage we may apply double ligatures to some of the varices of the leg and then remove them. Catgut ligatures have been passed through the skin and behind the varicose vein at the central and peripheral end of the varicosity, and then tied over a bit of rubber tubing. None of these operations are entirely free from danger from the thrombosis which must occur and may cause death, nor do they radically cure the disease. Of what use is it to cut off a few branches of the dilated venous system of the leg, for instance? In a short time other branches will become varicose. Experience accords with this theoretical reasoning; so do not operate for varices without some urgent reason.

Patients who can afford it may wear thin rubber bandages (*Martin's*); they are cheaper and more durable than elastic stockings, and may be worn even when there are varicose ulcers; these may be coated with a mixture of iodoform and mucilage, and the elastic bandage be applied not very tightly every morning and removed at night.

To diminish very extensive varicosities we may resort to a procedure recently employed, aiming at shrinkage of the tissue around the veins and consequently of the dilatations, which is far less dangerous than other operations. This is the subcutaneous injection of certain fluids (dilute alcohol, ergotine, etc.), not into the veins, but into the tissue around them; this induces local, sometimes severe inflammation, which, however, does not usually advance to formation of abscess, but only to cicatrization and shrinkage of the perivenous tissue; if these injections be repeated at various points for a long time, they may cause obliteration of the veins. This method is greatly praised by *Schwalbe* and *Englisch*.]

# CHAPTER XX.

## *TUMORS.*

### LECTURE XLIV.

**Definition of the Term Tumor.—General Anatomical Remarks; Polymorphism of Tissues.—Points of Origin of Tumors.—Limitation of the Development of Cells to Certain Types of Tissue.—Relation to the Generative Layers.—Mode of Growth.—Anatomical Metamorphosis of Tumors; their External Appearances.**

GENTLEMEN: To-day we enter on the difficult chapter that treats of tumors. The swellings of which we have hitherto spoken depended only on a few causes; they were due to abnormal collections of blood in and outside of the vessels, to infiltration of the tissue with serum, to its permeation with young cells (plastic infiltration), either separately or in combination. In contradistinction to these swellings, we now in the clinical sense of the term call new formations *swellings* or *tumors* when we suppose they are due to other causes than those of the inflammatory new formations, and have a growth which as a rule has no typical termination, but, as it were, goes on *ad infinitum;* besides, most of these growths are composed of tissue which is more highly organized than inflammatory neoplasia. Let us investigate this more accurately. At present you are only acquainted with that variety of new formation caused by inflammation; this is very uniform, not only in its mode of origin, but in its further development; its development might be interfered with by disintegration, drying up, breaking down into pus, etc.; it might proliferate excessively, but it was always in such a way as not to change its character; but, finally, if there existed no specially unfavorable local or general cause, and no vital organ was disturbed by the new formation, it subsided—it again became connective tissue; the inflammation terminated in cicatrization. Then, if the inflammation was superficial, there was development of epithelial or epidermis cells, the bony cicatrix ossified, new nerve-filaments formed in the nerve-cicatrix; in all these changes the development of new blood-vessels played an important part; still, as above said, the typical termination of the inflammation, whether it was acute or chronic, superficial or deep, was in the cicatrix.

Although connective tissue, nerve, and bone tumors, may exceptionally form from connective tissue, nerve, and bone cicatrices, still these constitute a very small part of the various tissue-formations found in tumors; forms the most varied and complicated, such as newly-formed glands, teeth, hair, etc., are occasionally to be found in the tumors; indeed, tissues are there seen which, as then arranged, never under other circumstances occur in the body or even during fœtal life. To enable you to form a correct idea of the anatomical characteristics of tumors, I will recall to your memory a few general laws from general pathology about the formation of new growths; in the large works on this subject by *Virchow* and *O. Weber* you may find very excellent and exhaustive representations of these conditions.

When a part of the body is abnormally enlarged, we make a distinction as to whether the enlargement is caused by an abnormal increase of volume of the different elements (*simple hypertrophy*) or by a formation of new elements, which are deposited between the old ones. This new formation may be analogous to the matrix, or mother-tissue (*homœoplastic*), or not (*heteroplastic*). The homœoplastic new formation proceeds either from simple division of the existing elements (thus a cartilage-cell by segmentation forms two, then four cartilage-cells); then it is called *hyperplastic* (numerical hypertrophy); or at first apparently indifferent, small, round cells form from the existing cellular elements, and from these a tissue analogous to the matrix is developed—homœoplastic new formation in the strict sense. Heteroplastic new formations always begin with the development of primary cell-tissue, so-called indifferent formative cells (granulation stage of tumors, *Virchow*), and from these develops the tissue heterologous to the matrix (as cartilage in the testicle, epidermis in the brain, etc.).

This nomenclature, proposed by *Virchow*, seemed perfectly suitable and natural in a purely anatomical point of view; and I can still accept it if the term of heteroplasia be limited, as will be hereafter stated, and if we dismiss the idea that homœoplastic is synonymous with benignant and heteroplastic with malignant. We must here add that there is every probability that *wandering cells* escaping from the vessels very materially aid in the formation of tumors, at least to the formation of tumors of the connective-tissue series. But, apart from this, we should err if we supposed that in the above nomenclature all cases of new formation, even considered in a purely anatomical point of view, could be easily labelled, ready to be placed away in a museum. The simple numerical hypertrophies and hyperplasiæ, although in some cases difficult to distinguish, are at least theoretically separable; the same way with those new formations which do not consist of similar, well-formed tissue-elements; a connective-tissue tumor occurring in connective tissue would always be termed homœoplastic; found in bone, brain, or the liver, it would be termed heteroplastic, etc. Well-developed alveolar cancerous tissue also usually presents no difficulty of classification, for it does not normally occur in any part of the body, it is everywhere heterologous. But what shall we say of the neoplasiæ which have no fully-developed normal or entirely abnormal form of tissue, but consist of elements that cannot be found elsewhere; what becomes of them? or, can any thing develop from them (indifferent formative cells, primary-cell tissue, granulation-tumors)? and where shall we place those neoplasiæ which are not completed tissue, but are evidently normal tissue in the stage of development? According to the above definition of heterology and homology, inflammatory new formation is at first heterologous everywhere; but the connective-tissue cicatrix developing from it subsequently becomes a homologous neoplasia in connective tissue; in muscle it almost always remains heterologous, the same way in the brain and in the bones, if it does not ossify. You see that here parts, which from their nature and mode of origin naturally belong together, are sundered by the anatomical nomenclature. But let us leave inflammatory neoplasiæ out of the question. Every tumor resulting from indifferent formative cells must exhibit a series of stages of development, if the cells are transformed to one or several sorts of tissue. Wherever they are grouped together, indifferent formative cells are heterologous; if a neoplasia show only such elements, we will let it pass for heterologous; but if it appear that a number of these cells have been transformed into spindle-cells, the question arises, Where does this neoplasia belong? Spindle-cells collected in groups are heteroplastic in all parts of the body; but these cells occur in fœtal connective tissue, fœtal muscles, and fœtal nerves; what would finally become of the spindle-cells of this tumor? if found in muscles, should not this tumor still be called homologous? On this point we can only decide arbitrarily; you may look at it from different points of view. Now, what shall we do with tumors that contain the most different complete and incomplete tissues? I will stop here, to avoid making you skeptical; it is my duty to help you learn, not to throw obstacles in your way.

As the enlargement of the individual elements (simple hypertrophy) cannot be observed, and the increase of the elements from themselves (hyperplasia) is an act often observed and constantly going on in physiological growth, it only remains to treat of the point of origin of the indifferent formative cells, and their further course. Here we find ourselves in the same position as in inflammation, only in regard to the development of tumors we unfortunately cannot make any experimental investigations. Formerly the proliferation of connective-tissue cells was not doubted, and these were assumed as the source for the development of most tumors. But most, possibly all, of these indifferent cells are wandering white blood-cells. There is little doubt that on this point there was formerly much error, conclusions having been too quickly drawn from the arrangement in groups, and the metamorphoses of the formative cells; nor can I claim to have escaped these errors. For instance, when in sarcoma we found small indifferent cells, with one, two, and then more nuclei near together (when between the filaments of the connective tissue, where the connective-tissue cells lie, we saw a small, then, near by, a large group of indifferent cells), the conclusion that the new groups of cells were derivatives from the connective-tissue cells seemed quite unprejudiced; also, that from these indifferent cells, larger multinucleated ones were constantly developed till the so-called giant-cells were arrived at. Knowing now that an infiltration of the tissue with small cells may depend on escape of white blood-cells from the vessels into the tissue; as before remarked, we also become doubtful about the origin of the indifferent formative cells in the tumors. Of late, especially in glandular and epithelial cancer, I usually seek in vain for proliferating connective-tissue cells, although the whole connective-tissue layer of these tumors is generally infiltrated with young cells. The deep obscurity which had surrounded the origin of young epithelial cells has only lately been cleared away. From the latest investigations we know that these cells increase by a sort of segmentation. I must here remind you of what was said about the regeneration of tissue in inflammation (Lecture XXII.). From *Arnold's* observations we may suppose that, in the development of tumors, protoplasm which has been fully changed to tissue may possibly pass into a granular condition, a nucleus may form in it, and it may then proliferate, and segmentation occur as it does in cells; in which case new tissue is first formed when the granular protoplasm has changed into cells; so that *Schwann's* law that "all tissues are formed from cells" is not broken, although there is a modification of the law that "every cell comes from a cell."

We have frequently spoken of *indifferent formative cells*, without having sufficiently defined this term. By these we mean the small, round cells which everywhere first appear after irritating the tissue, and with which we became acquainted in inflammatory new formations. Until within a few years I believed that these young cells were actually as indifferent as the primary segmentation-globules of the egg [vitelline spheres of *Dalton*], i. e., that any tissue might finally develop from them; and more especially I thought that not only all forms of connective-tissue substances (connective tissue, cartilage, bone), vessels, and nerves, but also epithelial tissues, glands, etc., could proceed from the derivatives of the connective-tissue cells. Against this still prevalent view *Thiersch*, in an excellent work on "epithelial cancer," has produced such proofs that I must entirely agree with him. As I propose returning to this point hereafter, when treating of cysts, glandular tumors, and epithelial cancers, I shall here merely point out the general outlines of my views. From the account of development you know that the body of the young embryo very early shows three different layers, so-called germ-layers. As soon as the division of the cellular embryonal elements into the three germ-layers is accomplished, all observers agree that each of these three germ-layers produces only a certain series of tissues. From the horny layer are formed the nerve-system, the epidermis, and their derivatives, the cutaneous glands, the sexual glands, the labyrinth of the ear, the lens; from the middle germ-layer are formed the connective substance, the muscles (?), the vascular system, the lymphatic glands, the spleen, the peripheral nerves (?); from the inferior or glandular layer are formed the epithelium of the intestinal canal, that of the lungs (?), all the secreting elements of the liver, pancreas, kidneys, etc. This is one of nature's laws, for whose discovery we are greatly indebted to *Remak*, *Reichert*, *Kolliker*, *Heis*, *Waldeyer*, and others, and which may probably be carried back into the composition of the ovum. In the whole subsequent course of development a derivative of one germ-layer never develops a tissue which was originally formed from another; in other words, if the division of the cellular embryonal plan has advanced to the three germ-layers, there are no more wholly indifferent cells, but all newly-formed cells developed from previous ones can only develop to tissues lying within the territory of the germ-layer whence they originate; cells originating from true genuine epithelium can never produce connective tissue, and true epithelium or glands can never come from the derivatives of connective-tissue cells. There is no reason for supposing that the natural law would be annulled if the cellular elements of the complete organism were excited to production by any irritation; the young brood can only develop to certain prescribed types of tissue, which depend on the embryonal origin of the mother-cells. When we have spoken, or in future speak, of indifferent cells, you must always limit the expression by the principles above developed. If we now return to the system of new formation developed by *Virchow*, according to our view there is no such thing as a true heteroplasia, for the germ-cells formed from the derivatives of one germ-layer can only develop differently within certain bounds; they can never become one of the types of tissue belonging to another germ-layer. From the great movements constantly being made in histogeny, any very absolute assertion is in danger of being obliged soon to submit to some modification; still I must repeat that it seems to me in the highest degree probable that a large part of the young cells escaping so extensively into the tissues during the development of tumors are movable, wandering, connective-tissue cells, that is, escaped white blood-cells. Nevertheless, I would not deny to the stable elements all participation in the new tissue formation. For instance, it has been proved of muscular filaments that their cells proliferate after irritation, by division of the nuclei, although this may not occur for some time (in rabbits about the end of the first week); the same is true of the nerves; the cartilage-cells also react on irritation, although not for some time. It is uncertain whence the wandering cells come (they are identical with white blood-cells and lymph-cells); probably their original source is from stable elements of the lymphatic glands and spleen; at all events, they must be regarded as elements of the middle germ-layer, and hence their powers of development must be regarded as limited to the tissues of this layer. Our times may look with pride at the progress of modern morphology, whose importance is proved by the very fact that it is so destructive to previous views and so fruitful in the most diverse directions.

When some investigators on this point assert that the conditions of embryonal development just given have no claim to pass as immutable laws of nature, but only serve as rules for the development of the more highly-organized animals, I must leave the embryologists to maintain the argument. But I would entirely deny the assertion that types of development which are recognized in embryology have no analogy in neoplasms which result from various irritations of developed tissue; for all modern histogeny is based on the principle that *development of pathological neoplasia is only a repetition of typical development of normal tissue*, which has been generally accepted since *Johannes Müller's* pioneer work on tumors. If we lost this principle, we should lose all our hold on this domain and fall back into the old chaos of parasites and pseudoplasms.

Let us now return to tumors. Their life and growth may vary greatly. In the first place, the diseased portion of tissue, the first tumor-nodule, may grow in itself, without new points of disease developing in its vicinity; in the midst of the tumor itself, from the cells collected at a circumscribed spot, new ones constantly form, with a tendency to develop in the same direction, predestined as it were for the type of development taken by the new formation. It was formerly supposed that the distention of the vessels was a very essential indication of inflammatory neoplasia; numerous researches in this direction have shown me that the enlargement and new formation of vessels in the development of the first tumor-nodules are not inferior to those in inflammation. It has not yet been proved that there is a softening of the capillary and venous walls, as in inflammation. The original focus of disease may also grow by new foci constantly forming in its immediate vicinity; an organ once diseased in this way is not only compressed by the tumor, and its elements separated, but it becomes more and more diseased, and so becomes infiltrated and destroyed by the tumor, and is finally transformed into it; for you have already seen that a neoplasia forms in normal tissue, the matrix ceases to grow, and is partly transformed into the new tissue, partly is destroyed. So in the first case we have an isolated focus of disease which, once existing, draws the material for its increase from its own cells; in the second case we have a continual extension of the foci of disease. The first variety, the to some extent pure *central* growth, is decidedly less unfavorable to the organ diseased than the latter, the *peripheral* growth, which, when it continues *ad infinitum*, must cause complete destruction of the organ, just as when an inflammation or inflammatory new formation continues progressive. A combination of these two modes of growth is the most unfavorable, but unfortunately is quite frequent. If we study the life of the tumor itself further, we find that the neoplastic tissue does not by any means remain stable, but is subject to some changes, such as are also seen in inflammation. From various causes, acute and chronic inflammations may develop in the tumors, i. e., with pain, swelling, and enlargement of the vessels; there is an infiltration of small cells into the tissue of the tumor, which may even lead to suppuration; this disease of a tumor is the more frequent the less its elements are organized to a stable vital tissue, especially the less its vascular system is regulated and fully organized. Tumors in which the cell-formation is so excessive and progresses so rapidly that the formation of vessels only follows up the growth of the tumor slowly are least capable of living; slight disturbances then suffice to impede the whole process of development, or, as they do not arrest it entirely, to cause destruction. We must examine somewhat more minutely the metamorphosis of the tissue of tumors in inflammations. They may come on in an acute or chronic manner; acute inflammations are on the whole rare, still they may be induced by injuries, blows, or contusions; this traumatic inflammation in vascular tumors rich in connective tissue may terminate in resolution with or without cicatricial contraction, but frequently they are followed by more or less extensive extravasations, gangrene, or suppuration. Chronic inflammations in tumors are far more frequent, both those characterized by production of inflammatory neoplasia, fungous ulcerations with great vascularization, and those marked by torpid ulceration. Caseous and fatty degeneration of the tissue and its breaking down into mucous fluid are not very unfrequent occurrences. In these processes of softening there are thrombosis and collateral dilatation of the vessels around the softening point, as in the transformation of a focus of inflammation to an abscess or to caseous matter. All these changes, by development and disease of the tumor, may so complicate its appearance as to render it sometimes difficult at once to tell correctly, in any given case, what was the original tissue of the tumor. Lastly, it sometimes happens that in the course of time tumors change their anatomical state; for instance, a connective-tissue tumor which had long continued in that state becomes softer by rapid proliferation of cells and greater vascularization; or, on the contrary, a soft tumor becomes hard from atrophy of the cells and cicatricial contraction of the connective tissue existing in the tumor. So you see what an amount of knowledge and experience is necessary merely to judge correctly in each case of these anatomical conditions, which form the basis of all our knowledge of tumors; indeed, we may occasionally be unable to give to the object we have examined a name by which it may be simply labeled in one of the regular groups; as regards the nomenclature of tumors which are composed of various tissues, we generally choose the name from the tissue that is present in the tumor in the largest amount.

It has been generally agreed to append *ὤμα* to the name of the affected tissue, to characterize a tumor histologically; as sarcoma, carcinoma, etc. There was no word *ὤμα* among the Greeks; it came from giving certain nouns the termination *οω* to make them verbs, as: *σάρξ*, flesh, *σαρκόω*, to make flesh; *καρκίνος*, cancer, *καρκινόω*, to make like cancer. The Greeks used the expression *σάρκωμα*, fleshy tumor, *καρκίνωμα*, cancer, ulcer (*Hippocrates*). Modern nomenclature has been developed from this, and has been carried out by *Virchow* with especial thoroughness. The old Grecian term for tumor in general is *ὄγκος*, bend, bending, bulk, mass, etc.; hence *Virchow* has termed the study of tumors "onkologie." The term *φύμα*, *φυτόν*, growth, also used by *Hippocrates*, is now rarely employed.

*Celsus* occasionally designated tumors in general as "struma," but glandular tumors on the neck were more especially meant. The English term "strumous" what we call "lymphatic, scrofulous." The Germans confine the term "struma" to tumors of the thyroid gland.

I have little to say about the external gross appearances of tumors. In most cases the growths are roundish nodules, more or less distinguishable, by sight and feeling, from the surrounding parts. This is not always accurate, however; tubercles also, at least in their smallest state, are bounded roundish bodies, which I should no more class among the growths than I should papules and pustules of the skin. In the skin also a distinctly-formed nodule may appear as a growth, just as an abscess may which also at first appears as a nodule. Still, as chronic inflammatory new formations on the surface also frequently appear in the form of *papillary proliferations* (tufts), a growth forming on the skin or mucous membrane may also assume the papillary form; even the surface of a tumor, or a newly-formed cavity containing fluid or pulp, may produce papillary proliferations. So you see that growths and inflammatory neoplasia are not accurately distinguishable by their purely external anatomical conditions.

There are a number of terms for different peculiarities of tumors, which are frequently used even now, although they do not always refer to any essential point. Thus, a tumor situated in a cavity, and attached by a pedicle, is called a *polypus;* so, we speak of nasal polypi, uterine polypi, etc., but must add the histological peculiarities (as fibrous, myxomatous, etc.). Growths that are ulcerated and project like a fungus are called *spongy*, or *fungous.* Formerly, if one wished to say that a tumor was very vascular, he used the word "hæmatodes," while to-day it is called "telangiectatic," or "cavernous." If a tumor was very firm or fibrous (not cartilaginous or bony) it was formerly called "scirrhous," which merely means "firm," and was applied to inflammatory new formations just as to cancer. A tumor was called *medullary* when it had the color and consistence of the brain, while its structure might be that of sarcoma, carcinoma, or lipoma. As tumors of this appearance are recognized as peculiarly malignant, the terms "medullary sarcoma," "medullary carcinoma,' have been applied to malignant tumors in general without regard to their structure. Some growths are colored—brown, yellowish, brownish black, bluish black; this pigmentation may be due to extravasations, or to specific cell-activiy. *Melanomata* or *melanoses* are rare, partly or entirely black or brownish-black tumors, with the structure of sarcoma or carcinoma, and usually of very bad prognosis. Formerly only these and similar terms, and comparisons to this or that tissue, were used; it is enough for you to know what they mean.

We must again return to the term "tumor." Pure anatomy should simply reject this term, for it acknowledges only simple or composite tissue-formations (organized neoplasia of *Rokitansky*); from a series of observations it can show how these structures develop, and what becomes of them; we shall not thus arrive at the term "tumor" in the sense in which we use it in pathology. Tumor, or growth, in the pathology of to-day, has a decidedly etiological and prognostic signification; as stated at the opening of this section, it is a neoplasm that has not started from the same causes as excite inflammation, but from others that are unknown or but vaguely suspected; the process in the organism (local or general) that produces tumors is generally considered different from inflammation; some regard the two processes as antagonistic to a certain extent (we shall not here discuss the correctness of this view). If in any given case we have to admit that factors which generally cause inflammation (traumatic, thermic, chemical irritation, etc.) have not caused the development of the tumor, the case seems so unusual that we are disposed to regard the growth as an unusual organism. This pathologic or physiological view, as I might term it, was not formerly maintained, but I do not think I err in stating that, consciously or unconsciously, it is held by most pathologists. All writers on tumors, as much as possible, avoid speaking on this point, as there is nothing more to say on it; for we do not know how or where we shall draw the dividing line between chronic inflammation and development of tumors. So it is not possible to have a purely anatomical idea of "tumors," any more than it is of the term "typhus;" to understand them we must make a compromise between etiology and pathological anatomy. The etiological expression, "the process by which tumors are developed," implies that the fate of the product or tumor will probably differ from that of the "inflammatory neoplasia;" hence we might say of tumors that they do not bear in themselves the conditions for a typical termination, as do the inflammatory neoplasiæ. I would not assert the inflammatory process is at all the opposite of that by which tumors are developed; on the contrary, I believe that observation teaches that, in some cases, the two processes correspond, especially in some forms of chronic inflammation and sarcoma, while, on the other hand, acute metritis and fibroid of the uterus are far enough apart, etiologically and anatomically. The idea that the development of tumors has certain specific causes, both in or external to the organism, is little disputed; and, when it is, it is hardly in earnest. *Virchow* asserts that the development of tumors may start from an increase of the inflammatory diathesis; thus, polypi of the mucous membranes result from long-continued catarrh; syphilis induces, first, inflammations; then, tumors. I would incidentally remark that I do not consider any product of syphilis a tumor; a gummy nodule or a caseous nodule, caused by syphilis, either heals by reabsorption, or, after being slit up, by suppurating and cicatrizing, while in an incised tumor this is exceedingly rare. *H. Meckle von Hemsbach* advanced the opposite idea, e. g., he says enchondroma of the finger is the mildest expression of a scrofulous diathesis. If we compare the products of inflammation with the histologically more developed tumors, it must be acknowledged that, as being the more slowly developed neoplasiæ, tumors are probably due to a feebler local irritation, more allied to normal growth. All these considerations apply only to true growths. In what follows we shall treat of these alone. When *Virchow* classes encapsulated extravasations of blood and dropsies of serous sacs among the tumors, he goes beyond our present views.

[Heteroplastic neoplasia, in relation to the part on which they are located, may be *heterochronic;* i. e., in an adult a tissue may develop, which in fetal life was physiological, but which does not normally occur in extra-uterine life; or they may be *heterotopic;* i. e., a tissue analogous to some physiological type comes where it is never found normally. An example of this is the development in an adult of mucous tissue, like that which in the fœtus is normal, *Wharton's* gelatine (heterochronic neoplasia), or the formation of cartilage in the parotid gland (heterotopic).

In the change of protoplasm to cells spoken of (page 658), the "proliferation" (to use a current term) of granular protoplasm, according to *Nedopil's* investigations, has an analogue in the physiological development of the laminated lingual epithelium in man. In the embryo and the newly-born, the lower layer, bordering on the vessels, consists of a mass of granular protoplasm, with no cell-boundaries, but with nuclei freely proliferating; this is the epithelium-forming layer, which toward the surface forms nuclei surrounded by protoplasm, i. e., sharply-bounded cells. According to *Nedopil*, the development of lingual carcinoma takes place in the same way from the epithelium-forming layer, which to some extent assumes its embryonal condition, as if formed of homogeneous protoplasm.

In continuation of what was said (page 559), more recent studies of the history of development, which were first made on the lower animals, have given very important knowledge, which we will briefly consider here as far as it concerns us. Instead of the three germ-layers, we at present distinguish the ectoblast, the entoblast, and the parietal and visceral layers of the mesoblast. Ectoblast and entoblast are first formed, and are the two primary limiting layers of the organism. The middle germ-layers, the parietal and visceral mesoblast, always come later, and form from eversion or inversion of the entoblast; besides these germ-layers, and distinct from them, are *embryonal cells* which come from a union of the original elements, and are called mesenchymal germs, or original cells of the mesenchym. They serve to develop between the epithelial limiting layers a special tissue, the so-called mesenchym, from which are derived the numerous forms of connective tissue, muscle-cells, nerve-tissue, blood-vessels, and lastly the blood itself.

Adult animals have the outer and inner limiting layer of the body, the ectoderm and entoderm, which, developing from ectoblast and entoblast, have retained the original position. But between them, as mesoderm, have been pushed all those tissues and organs that have been developed from mesenchymal germs, the mesoblast, or directly from one of the germ-layers.

From this account, based on *Hertwig's*, it is evident that the former separation of different tissues, according to their development from the three germ-layers, cannot be considered exact. Connective tissue is not so genetically distinct from true epithelial tissue, as the former proceeds from mesenchymal germs which have come from the two original epithelial layers. This is very important in studying the development of tumors. In physiology also there are many things which do not well accord with the former representation of the germinal layer theory. The recently published investigations of *Swaen* and *Masquelin*, on the development of the maternal part of the placenta in rabbits, have shown that the epithelial-like cells of the decidua materna come from the proliferating connective-tissue cells of the walls of the vessels as well as from the true epithelial cells of the uterine mucous membrane; also that protoplasm masses, originating from glandular epithelium, enter between the connective-tissue cells of the walls of the vessels and combine with the intercellular substance of the latter; lastly, that true epithelial cells of the uterine mucous membrane, and epithelium of the uterine glands, after various metamorphoses, form hæmoglobine, the substance of the red corpuscles, which may be found in the protoplasm of these cells as well as in the glands, as a product of proliferating cells.

Old observations may be regarded as incorrect, but there are many recent pathological observations on development of tumors, especially of carcinomata, which speak decidedly against a sharp division of tissues, particularly of epithelial, connective, and muscular tissues. For my part, I think that there are indifferent cells in the perfect organism, and that under certain circumstances any living cell may return to its former state, where it was an indifferent formative cell; as a type of this latter, we have white blood-corpuscles (*Recklinghausen's* wandering cells). All cells from the connective-tissue series, the endothelium of serous membranes, and, above all, the fixed connective-tissue cells of the tissues, may be regarded as metamorphosed wandering cells or white blood-corpuscles; and it is easy to suppose that a fixed cell in the connective tissue may proliferate under certain circumstances, and produce a young cell which shall have all the characters of an indifferent formative cell—that is, of a white blood-corpuscle. But, on the other hand, we can describe no difference between young formative cells; from which the so-called heterologous neoplasia develop, and the derivatives of proliferating connective-tissue cells. All tissues, with few exceptions, may produce such young cells; indeed, it seems as if even a complete cell were not necessary for this; any protoplasm may develop a nucleus which divides and causes division of the cell. So it appears that all cells having the properties of living protoplasm may produce young cells of more or less indifferent character, which equal white blood-cells, and like these may pass through several stages of development. Hence it is not strange that various observers have witnessed the proliferation of the endothelial cells of the vessels and their transmutation to epithelial cancer-cells; and in the same way the production of the same cells by proliferating connective-tissue corpuscles. Endothelial and connective-tissue cells are really only fixed wandering cells, or white blood- or lymph-corpuscles. But where these cells enter the blood is uncertain; probably they originate from stabile elements of the lymphatic glands and spleen. You see that there is a continuous circulation in the development, even physiologically. There is nothing strange in supposing that, in the development of tumors, tissue-cells should return to the point where they stood before the change in the germ-layers and mesenchymatous germs. Then, strictly speaking, there were only epithelial cells which composed the ectoblast and entoblast; hence all the material for later tissues originates from one source, primitive epithelial cells; just as these come from ovum-cells fecundated by the sperm-cells, that is, from the copulation of two epithelial elements.

Our age may look with pride on the advances of modern morphology, whose importance is shown by its disturbance of former views, and by its contributions in various directions.

Tumors all pass through the same course; blood-vessels form from those already existing, and keep pace with the formation of actual tumor-elements; it is not, as was formerly supposed, first a tumor formed like a parasite, with vessels subsequently growing into it from the periphery. It has not yet been determined whether the wandering of colorless blood-corpuscles is a part of this change.]

## LECTURE XLV.

Etiology of Tumors; Miasmatic Influence.—Specific Infection.—Specific Reaction of the Irritated Tissues; its Cause is always constitutional.—Internal Irritations; Hypotheses as to the Character and Mode of the Irritant Action.—Course and Prognosis: Solitary, Multiple, Infectious Tumors.—Dyscrasia.—Treatment.—Principles of the Classification of Tumors.

Let us now go more minutely into the etiology of tumors. Here we should propose to find the differences and points of resemblance between the processes causing inflammatory neoplasiæ and tumors. Let us start with the causes of inflammation, and compare them with those of tumors. Many acute inflammatory processes (exanthemata, typhus, etc.), and some chronic ones (intermittents, scorbutus, etc.), are due to miasmata and contagions, which enter the body from without. I do not know any acute miasmatic tumors; but goitre must be considered as a chronic endemic-miasmatic tumor; goitre cannot be regarded as a product of inflammation, as it never spontaneously retrogrades, suppurates, or shrinks up into a cicatrix; the cause is a specific external one, to which every one, especially the young, is occasionally exposed, who comes into a country where goitre is endemic; all are not equally disposed to it, there may be an hereditary tendency; infection probably occurs through the blood; at least, we cannot well imagine how the thyroid gland should be infected by local infection. Hence goitre is probably the local expression of a general infection, which occasionally evinces itself in the whole nutritive state, especially in anomalous development of the skeleton and its results (cretinism). We may also consider leontiasis and Oriental elephantiasis as chronic miasmatic infections, in which large masses of nodular fibrous tumors form in the skin on different parts of the body; still, I acknowledge that this is disputed territory, and that reasons may be advanced for classing these among the chronic inflammatory diseases, instead of among tumors. As regards *local infection*, or the transfer of fixed contagions from without, we know that inflammations of various kinds may be thus induced. By putrid substances *only* inflammations are induced; here I class, also, the so-called "dissecting tubercle," which I cannot consider as a tumor, because it disappears spontaneously, as soon as new infection ceases to occur. Inflammation is excited by inoculation with pus; the character of the pus determines the specific nature of the inflammation; pus may also excite a constitutional disease, which again may evince itself by multiple localized processes, as in syphilis. Can tumors be induced by inoculation with the juices of tumors, or with small portions of them? This is a disputed point; I consider it possible, but not proved; the difficulty of coming to a decision lies in the fact that it is not allowable to make such experiments on men. When such experiments often fail on the lower animals, it only shows that tumors from man are not transferable to them; tumors from beasts must be inoculated on beasts of like species; a few such experiments have been made by *Doutrelepond*, in which the inoculations of carcinoma from dogs on dogs had no effect.

[Transfer of medullary carcinoma from one dog to another is said to have succeeded twice; after transplantation of small portions of a tumor in two dogs, there were produced nodules in the subcutaneous cellular tissue, of which one ulcerated, and was complicated with infection of the lymphatic glands; the structure of the nodules corresponded with that of the original tumor. Numerous experiments have proved that parts of tumors as well as pieces of tissue of very young animals may be transplanted to other living beings, but they always die after a time and never attain a tumor-like growth. And in our literature there is no authentic case of a physician infecting himself while removing a tumor and acquiring the same kind of a tumor; while there are many instances of the transfer of syphilis by instruments, etc.

It has been shown that leprosy, a general disease endemic in Northern Europe, particularly in Norway and Sweden, characterized by formation of nodules, may be transferred by inoculation. According to recent investigations of *Armauer*, *Hansen*, and others, this affection is due to a specific bacillus lepræ. Strictly speaking, we cannot regard leprous nodules as tumors any more than we could syphilitic gummata.]

At all events, we cannot induce a tumor by inoculating with pus, which again seems to show the specific difference of the products. Perhaps some pathologists may here answer that "molluscum contagiosum" is an example of tumor-juice or constituents of tumors being inoculable on other persons. But the propriety of classing molluscum contagiosum among tumors is disputed, and instances of contagion are rare. The most striking proof of the distinctness of inflammatory products and tumors is offered by observation of the local and general infection, which we have innumerable opportunities of making. We have previously said a good deal about progressive and secondary inflammation of acute lymphangitis, which is always secondary (deuteropathic, *Virchow*), of the secondary acute and chronic swellings of the lymphatic glands in acute and chronic inflammations, especially of the extremities; I then told you that I considered it more probable that cellular elements from the focus of inflammation passed into the lymphatic glands, and, by their specific phlogogenous action, induced inflammation in the glands, which were analogous to the primary peripheral inflammations; tumors never develop through such local infections from inflammatory foci; if the primary inflammatory focus be removed, the swellings of the lymphatic glands also disappear. Similar infectious peculiarities also occur in many tumors, especially those which, like the inflammatory neoplasia, are very rich in cells; not only may the immediate vicinity be infected, and numerous new foci be formed immediately around the first nodule, but very often the lymphatic glands are also affected, and secondary tumors form in them, which have the same peculiarities as the primary; nor are they any more apt to disappear spontaneously than the primary, even when the latter is removed; on the contrary, similar tumors then frequently appear in other quite remote parts of the body—*metastatic* tumors. Here you again have the analogy with the course of infection in inflammation, as well as the specific distinction, for metastatic growths never result from phlogistic infection, any more than metastatic abscesses in internal organs do from infection by a tumor. Infection is not common to all tumors, although, unfortunately, the majority are infectious; these are called *malignant*, in contradistinction to the *benign*, or non-infectious. It is difficult to say on what this difference is based; it is probably partly due to the nature and specific character of the element, in their easy mobility, and in the fact that, like the seed of some of the lower plants, they find almost everywhere soil suited for their development, and can grow in most tissues of the body; probably it is also partly due to the fact that the conditions are more or less favorable to the entrance of the elements of the tumor into the lymph or blood-vessels; for instance, it is remarkable that frequently very soft tumors (medullary sarcoma) consisting almost entirely of cells, when surrounded by a firm connective-tissue capsule, cause no infection of the lymphatic glands; we notice the same thing in some large encapsulated abscesses. In regard to metastatic abscesses, I have already told you that, according to my view, they are due to embolism; we should have to seek another explanation of diffuse metastatic inflammations. Diffuse metastatic tumors are very rare; I should apply this term only to a few forms of pleural and peritoneal carcinoma or sarcoma. As regards the mode of origin of metastatic tumors, the actual course of the infection, from analogy, it seems very probable that they, like the secondary tumors of the lymphatic glands, are induced by seed from the primary tumors, or from the tumors in the lymphatic glands. I acknowledge I am much inclined to this supposition. Although I could not formerly believe that the cells from a focus of inflammation or from a tumor could be as independent as thistle-down, still, I think that, with our present knowledge about the independent life of pathologically-neoplastic cells, there can be no doubt of the possibility of such a process. Quite recently an observation has been published which is a new proof of the great independence of the cells of the rete Malpighii; I mean the epidermis transplantation of *Reverdin*, which has been so often mentioned. This renders it even more probable than formerly that detached cellular elements of a neoplasm, carried to some other part of the body by the blood or other fluids, may there continue its growth. Although, on the first development of a tumor, as on the occurrence of an inflammatory new formation, the lymphatic vessels are partly closed, and may be filled with cells, still, subsequently, from compression, lymphatic and vascular thrombi may form, into which specific tumor-elements enter, and small particles of thrombi, which might form during the softening of the tumor, may enter the circulation, become attached at different places, and form new tumors. In veins, the formation of such thrombi filled with specific tumor-elements has actually been observed, and, at the same time, analogous tumors have been found in the branches of the pulmonary artery. It is important to remember that metastatic tumors, like metastatic abscesses, are chiefly found in the lungs and liver, except in cases where direct metastasis is very easy, as in pleural tumors, which develop as a result of primary mammary tumors, as in hepatic tumors found with those of the intestines or stomach; in these cases a direct wandering of tissue-elements through the lymphatic vessels is very possible. On this point there is still much room for investigation, which, I think, will meet great results. As we have already seen, the products of acute inflammation mostly have a pyrogenous action; those of chronic inflammation lack this peculiarity almost as much as do those of tumors; fever only occurs in the latter when there is disintegration of the neoplasia, and the products of the disintegration enter the circulation; more frequently, infection with such excreted matters shows itself in chronic inflammation in tumors by a general cachectic state, especially by disturbance of the general nutrition.

If we consider what has been said about the contagiousness of tumors, we see that there is some probability of their transfer from one person to another, though it is not proved; but there can be no doubt that the lymphatic glands and other organs may be gradually infected by various kinds of tumors.

As regards the effect of *taking cold* locally and generally as a cause of inflammation, there are no observations which would justify us in referring tumors to a similar cause. I do not know that any one has ever asserted and proved that tumors result from catching cold.

Views vary greatly about *mechanical* and *chemical influences* as causes of tumors. Various as the irritations may be, and much as they have been experimented with, in no single case has a tumor been caused intentionally by mechanical or chemical irritation; inflammatory new formations thus developed do not long outlast the external irritation. Wherever and however we apply such mechanical and chemical irritants, we only induce inflammations; if there be any specific mechanical and chemical irritation (I mean one acting on the organism from without, not starting from the tumor), i. e., one from whose action a tumor *must* develop, it is at present unknown. Then the question arises whether there are any reasons which render it absolutely necessary to assume such mechanical and chemical irritation outside of the organism. I cannot agree to this. It is true there are many cases where a tumor forms after a blow, kick, or injury, but the number of such cases is very small in proportion to those where, after similar causes, there is acute traumatic inflammation, with a typical course, or, if the irritation be continued, chronic inflammation also with typical course. We must regard this also as a rule: if a porter gets a thickening of the skin, with new mucous bursa under it, on the spinous process, or if he gets an ulcer at the same point, it is to some extent a normal result, they are products of a chronic inflammatory irritation, and disappear as soon as the irritation ceases; but if from the same causes a person gets a fatty tumor, which does not disappear, but even continues to grow when the irritation ceases, we cannot here regard the irritation as specific, but must seek the peculiarity in the affected part. Previously in general and local infections we recognized the specific effects of irritation, now we must also acknowledge that there is a specific, qualitative, abnormal reaction of the tissue. *Virchow* and *O. Weber* especially have maintained that external irritation always plays an important *rôle* in the development of tumors; this follows undoubtedly from the fact that primary tumors are most frequent at points most subject to external irritation. Statistics show that the most frequent seat of tumors is the stomach, then the portio vaginalis uteri, then face and lips, then the mammary glands, rectum, etc. But the reason for the development of tumors, and not of chronic inflammation in such cases, must be a specific disposition of these parts in certain persons. Individuals who drink much spirits usually have gastric catarrh; if, among one thousand topers, one or even ten, instead of catarrh, had cancer of the stomach, he should be considered as an abnormal subject, when compared with the mass who do not have it. Up to this point I agree entirely with *Virchow*, who speaks as follows: "Although I cannot tell in what particular way an irritation must occur, to induce a tumor in some given case, while in another case, perhaps under apparently similar circumstances, it merely excites simple inflammation, still I have communicated a series of facts which teach that, in the anatomical composition of different parts, certain continuous disturbances may exist which interfere with the occurrence of regulating processes, and which, from an irritation that at another spot would have induced a simple inflammation, excite an irritation from which the specific tumor is developed." Among facts "which teach that, in the anatomical composition of different parts, certain continuous disturbances may exist" which dispose to development of tumors, *Virchow* mentions *advanced age.* It is perfectly true that certain forms of tumors are very frequently found on particular parts of the body in old persons, e. g., cancer of the lip. *Thiersch* calls attention to the fact that in the lips of old men the connective tissue is often so much atrophied that the epithelial tissues (sebaceous, sweat, and mucous glands, hair-follicles, etc.) become very prominent, and, as it were, receive the preponderance of nutrition; that hence irritation shows itself chiefly in the proliferation of these epithelial formations, and that this explains the frequent occurrence of epithelial cancer in the lips of old men. I fully recognize the shrewd combination of these observations, but I must add that *advanced age is just as much a general as a local peculiarity of the body.* It is also stated by *Virchow* that places which have been the seat of an inflammatory disease, which has left the part weakened, also cicatrices, furnish foci for the development of tumors. This is undoubtedly true; but if we compare the innumerable cases where simple chronic inflammation occurs in parts that have been acutely diseased, and where simple ulceration occurs in cicatrices, the cases in which tumors occur at such points appear very small, and it must be acknowledged that in these few cases we may assume a *specific predisposition* which leads to formation of tumors. The same holds good for the fact that tumors are particularly apt to form in organs which complete their formation and development late in life; here *Virchow* classes the articular ends of the bone (which, however, are the seat of tumors much more rarely than of chronic inflammations), the mammary glands, the uterus, ovaries, testicles, etc. While fully recognizing the exercise of observation and brilliant ideas by which it is attempted to prove the purely local disposition to development of tumors, I cannot consider the proof as at all convincing, but remain of the opinion *that there is just as much a specific predisposition to the development of tumors as there is to chronic inflammations, with proliferation of the inflammatory new formation, with suppuration, with caseous degeneration, etc.*

To what has just been said we must add that we cannot always detect a local external irritation when a tumor is developed any more than we can always do so in local disease in a scrofulous patient. While referring you to what has been said on the etiology of chronic inflammations, I would remark that in regard to primary tumors we may assume in many cases that there are also specific, so-called internal irritations developing in the body itself. Most pathologists agree to this, but they consider the mode of origin and development of such irritations as being different. *Virchow* teaches that the local disease must have a local cause, and assumes that at the point of disease there are certain local conditions of debility. If this were so, we should have to assume a specific local debility for the most different disturbances of nutrition and for formation of tumors. *Rindfleisch* speaks very decidedly of internal irritation as follows: "By the change of substance in the tissues, certain excretive substances are constantly being formed, which must gradually be passed off from the tissues and organs in which they form, as well as from the fluids of the body at large, in order that the life of the individual may be undisturbed. These bodies have their chemical position between the organopoietic bodies on the one hand and the excreted matter of the kidneys, skin, and lungs, on the other; thus they fall into the great gap that exists in organic chemistry at this point; they are different for the different tissues, and on this difference depends the variety of pathological new formations. If they are transformed and excreted normally they collect first at the point of their origin, then in the fluids of the body, and this collection is the immediate cause for the excitement of that progressive process which begins with multiplication of cells in the connective tissue, and ends with the development of tubercles, cancer, cancroid, fibroids, lipomata, etc." I can entirely agree with this hypothesis, but must add that it seems an error to suppose that we here speak chiefly of local processes. The production of bile and urine is also a local process; for them to be produced in such quantities and of such a quality as they are depends not only on the glandular organs, but on the entire organism to such an extent that we must seek the original causes of the secretion of urine and bile not only in the blood, but even more remotely, even in peculiarities of origin, as far back as Adam, if you please. In the same way, I think that the original causes for the local requirements for the development of tumors must be sought in specific peculiarities of the individual organism; in the same way we speak of a scrofulous or tuberculous person, meaning the pathological race, as it were, to which the individual belongs.

I must lastly add that the supposition that the cause of disease, the irritation inducing the tumor, develops locally, where the tumor afterward forms, is as purely hypothetical as any that has yet been advanced. Let us take arthritis as an analogy: *Zaleski* induced the most typical arthritis in a goose by ligating the ureters; an articular disease resulting from disturbance of the function of the kidneys. Possibly tumors might just as well develop in any tissue from disturbance of the hepatic function! Very many things are possible. We know nothing certain on this point, and move entirely in hypotheses. For my part, I find it just as allowable to assume a diathesis here, as in scrofula, arthritis, etc.; that, partly from unknown, partly from known causes of general nutrition and ordinary conditions of life, abnormal matters proceed, which have a specific irritant action on this or that part of the body, analogous to that of certain drugs. Lastly, if to this we add that the diathesis for production of tumor is hereditary, although not to such an extent as the chronic inflammatory diathesis, the doctrine of weakness localized in certain systems of tissue, or certain parts of the body, seems entirely untenable. There is certainly a local cause for the members of one family having large noses; in proportion to the face, they have grown larger than in other men, still the large nose of the father cannot descend directly to the son, it can only be inherited from the father through the spermatozoa, and there the original cause is to be sought; all peculiarities that descend by inheritance are unquestionably to be termed constitutional.

I have now occupied you some time with reflections which some of you may consider very tedious; they will ask me, Of what use are these things in practice? Then, unfortunately, I must acknowledge that practice pays little attention to them, because they are so hypothetical. Those of you to whom such ideas as we have just spoken of do not occur, I advise to pay no further attention to them; not to be *obliged* to speculate as to the final causes of things is, in a certain sense, an enviable quality.

For convenience, let us comprise, in a few short propositions, what we have said regarding the etiology.

Tumors, like inflammatory neoplasiæ, result from irritation of the tissue; the difference in the causes lies: 1. In the specific quality of the irritation. Infection of healthy tissue about a tumor, neighboring lymphatic glands, etc., is considered sufficient proof of this. It is supposed that, under some unknown circumstances, this specific irritant may be formed locally (*Rindfleisch*). I think that, partly as a result of hereditary predisposition, partly from a developed tendency, that is, where there is a diathesis, we may imagine the formation of materials in the fluids of the body, which shall have a specific irritant action on one or other tissue. 2. Any, usually an inflammatory, irritation may excite a tumor, if the irritated tissue is specifically disposed for the development of growths. *Virchow*, *O. Weber*, *Rindfleisch*, and others, assume that such specific peculiarities are entirely local and limited to an accidentally irritated part of the body, or to a certain system (bones, skin, muscle, nerves, etc.). I cannot imagine the localization of such specific peculiarities; hence, even with this hypothesis, it seems probable that the apparent local specific peculiarities are due to the intimate relations of the entire organism.

From this representation you may see that the different views only differ in the purely hypothetical part. If I entered into the subject more fully than seemed necessary for these lectures, it was because this very important branch of general pathology has lately been so exhaustively and excellently treated of by *Virchow*, *O. Weber*, *Rindfleisch*, *Lücke*, *Thiersch*, *Klebs*, *Waldeyer*, and others, that I considered it necessary to develop more fully those parts of my views where I differed from these authors, whose excellent writings I cannot too strongly recommend for your study.

In regard to the *prognosis* and *course* of tumors, from what has been said you may infer: 1. That they seldom recover spontaneously, nor are they accessible to medicines; and, 2. That they are partly infectious, partly not so. The latter point is particularly striking to unprejudiced observation. There are some tumors which do not return after extirpation, and others that not only return in the cicatrix, but come in the neighboring lymphatic glands and also in internal organs, as already remarked. The former have for ages been called *benignant*, the latter *malignant* or *cancerous*. This observation is so simple that it would seem merely necessary to study exactly the peculiarities of one or other form of tumor, to arrive at an accurate prognosis. But accurate clinical and anatomical study did not lead to this desired simple result of this dualism, but it showed that the latter did not exist, that the conditions were more complicated. After an exhaustive anatomical study and description of benignant and malignant growths, they were examined under the microscope and in the retort; it was thought that the characteristic marks had been found now in one point now in another, and soon one discovery after another proved erroneous: it was thus shown that an antithesis of absolute malignancy and benignancy did not exist in the sense meant, and that it was necessary to distinguish not only solitary, multiple, and infectious tumors, but that a scale must also be made in the grade of infectiousness. We must investigate this more closely. We call a tumor *solitary* when only one occurs in the body and causes purely local symptoms; they are usually growths consisting of any fully-developed tissue—fibroma, chondroma, osteoma, etc. We speak of *multiple* tumors when a series of similarly-organized growths occur only in one certain system of tissue; for instance, when numerous chondromata occur only on bones, or numerous lipomata only in the subcutaneous cellular tissue, or many fibromata only in the skin, etc. As generally acknowledged, there is at the same time a predisposition, which *Virchow* regards as purely local, but which, as already stated, I must consider constitutional. In general, we may say that all sorts of tumors may occur as solitary or multiple, although the latter is very rare in some forms of tumors. We apply the term *infectious* to a tumor which not only grows into the parts around it, infiltrating them and thus constantly growing by apposition of new foci, but which may also infect the next lymphatic glands and finally other organs. In this respect there are very great differences: in some tumors the infection extends regularly only to the next lymphatic glands (carcinoma of the lips and face); in other cases from that point it extends farther, especially to internal organs (carcinoma of the breast); lastly, infection of the entire body with metastatic tumors, without infections of the lymphatic glands, sometimes occurs (some forms of sarcoma). Moreover, the rapidity with which infection follows, varies greatly. If we examine the conditions under which infectious tumors develop, and their anatomical structure, we shall see that they occur especially in advanced age, about equally in men and women, and particularly often in certain organs; that the age of childhood is disposed to infectious growths, especially to malignant sarcomata, while in youth and the first years of adult age very few tumors of any kind, and especially few malignant tumors, develop. Mode of life, good or bad food, poverty, riches, character, nationality, and cultivation, appear to have no special influence on the development of tumors generally; nor can we recognize any specific influence of these powers on infectious tumors. The study of the anatomical structure of tumors has been pursued with great zeal of late, and it appears that a large number of malignant growths have characteristic macroscopic and microscopic peculiarities, but that a correct prognosis cannot always be based on them; in general we may say that they are usually very vascular tissue formations, disposed to ulceration, and in their course proving to be infectious. As it is most probable that the infection results from the locomotion of specific tumor-elements, some of the factors relative to reabsorption may here have some effect. The quantity of blood and lymphatic vessels in the tumor and its immediate vicinity, the conditions influencing opening and closure of these passages, and the activity of the circulation generally, are to be considered.

Infectious tumors are usually at first solitary, very seldom multiple in the sense above indicated. Tumors that are multiple from the start are rarely infectious. When we use the terms dangerous, malignant, and infectious, as synonymous, we do so without regard to the locality where the tumors are developed. A solitary benignant tumor in the brain is always malignant, from its locality; an infectious tumor at the same point possibly never goes beyond local infection, as it soon proves fatal. All these things are to be carefully weighed, if we would obtain clear ideas on these points.

Tumors are not always to be termed infectious (malignant, cancerous) because of a return at the point of operation. In this case it is very important to decide whether the recurring tumor has started from portions of the original tumor, that have been left at the time of operation (continuous recurrence, *Thiersch*), or, possibly years after a perfect operation, a new tumor has occurred from similar causes in the cicatrix or in its vicinity (regional recurrence). If the point of operation remains free, and, after the operation, swellings of the lymphatic glands, of the same nature as the extirpated tumor, appear, or if, under similar circumstances, without swelling of the lymphatic glands, growths occur in other organs, it may be considered certain that these lymphatic glands and other organs were already infected at the time of operation, although this may not have been susceptible of proof on examination.

When a person is infected from a tumor, we term it a *dyscrasia*, just as we do when one is infected from a focus of inflammation. In such persons foreign materials circulate in the fluids of the body, inducing in them a pathological condition. In infectious tumors this dyscrasia displays itself by general disturbance of the nutrition—emaciation, marasmus; how soon and how extensively this shall occur depends very essentially on the seat of the tumor and its peculiarities (softening, becoming gangrenous, ulceration, bleeding, etc.) as well as on the strength and age of the patient.

About the treatment of tumors in general I shall here merely mention that they are only curable by removal from the body, whether by the knife, ligature, écraseur, caustic, or any other means. The removal of intense and rapidly-infecting tumors is usually merely a means of prolonging life or of alleviating the sufferings of the patient; tumors that cannot be operated on we can only treat symptomatically, to ease the patient. I shall speak of the indications for operating when treating of the different forms of tumors.

Now, when passing to the consideration of the different forms of tumors, we shrink from the mass of material before us. We require a leading principle to enable us to arrange the various forms of tumors which differ so much anatomically and clinically, and to consider them in their relations to each other and to the organism at large. The principles on which tumors have been classed have for ages been just as different as those on which diseases generally have been and are still divided. None of the classifications of disease proposed so far have held their place long. Medicine is now taught in various groups of smaller systems, and the principles for forming such groups are chosen for various reasons. Before pathological anatomy was developed, some prominent symptom was taken; hence we still have in medicine the terms icterus, apoplexy, etc., to denote certain diseases; in the same way, as you know, we have tumors designated "polypus, scirrhus, lupus, fungus, carcinoma," etc. As soon as the symptoms icterus and apoplexy were analyzed and found to depend on very different anatomical causes, these terms were banished and replaced by others denoting the anatomical condition. The pathologico-anatomical arrangement of disease, as proposed by *Rokitansky*, for instance, is undoubtedly scientific, as is the system of general pathology of *Virchow;* still, neither of them is accepted without reserve by clinical teachers. It was desired to divide diseases according to their peculiar nature and cause; but *Schönbein's* attempt to found a system with this idea failed, for our knowledge of the causes and nature of disease is not sufficient fully to carry out the plan. What, then, is to be done? Practical medicine and surgery start partly from the anatomical system, consider this as generally known, and use it for subdividing more extended descriptions of disease founded on an etiological, prognostic, symptomatological, or physiological basis. It would certainly not be unscientific even now to write a monograph on icterus or apoplexy—then the anatomical conditions would come in the second rank; pathological anatomy is used as any other aid to science, as chemistry, physics, etc.; we always try to bear in mind that the object in fathoming the whole process of disease lies not in simply fathoming the morphological conditions; it is desirable to understand not only the anatomical change, but also the mode and causes of the physiological disturbances. It would be decidedly unscientific in typhus, even if a number of palpable changes were found, to admit nothing except the peculiar intestinal inflammation; we may regard this as something of the past. Could we group all diseases from an etiological point of view, it would be an immense advance; then pathological physiology would take the place of pathological morphology, while with our present knowledge we are quite proud if we accurately recognize the morphological development of the morbid product, for we can then say that we know at least one important factor of the pathological process. In fact, we know no more about normal development; it will be long before we understand the physiology of the growing fœtus.

After these considerations, we may not be any more particular about the classification of tumors than we are in the other diseases; we must see that there will be a difference according as we choose etiology, symptomatology, prognosis, or anatomy, as the principle for division. Formerly, surgeons preferred classing tumors according to the prognosis of the individual forms, into malignant and benignant, and adding a few subdivisions according to the appearance or consistence of the tumor or the looks of its cut surface. This was enough as long as observations on these subjects were made in the gross, and the surgeon made no great claims in prognosis. But the more accurate the observations at the bedside, and the more varied the forms in which the neoplastic tissue appeared under the microscope, the more impossible it became to make the anatomical peculiarities of tumors agree with the old views of malignancy and benignancy. While now most surgeons and pathological anatomists gave up the idea of letting the prognosis play a part in the classification, and since *Johannes Müller's* works on this subject turned their attention to working out the finer anatomy and developmental layers of the pseudo-plasms, I still made some attempts to retain the clinically-prominent symptoms of benignancy and malignancy in a more enlarged form, as a basis for the classification of tumors, and under these to arrange the modern acquisitions of pathological histology. Either I did not find the correct form and expressions for my ideas, or the task I tried was impossible, for I remained alone with my ideas on this subject, and have abandoned them. Although I am still of the opinion that we should not cease seeking for a physiological (etiological-prognostic, clinical), recognition of the process on which the formation of tumors depends, and although I should even now esteem a division of tumors on physiological-genetic principles more highly than one on anatomical-genetic principles (which was *Virchow's* idea in his wonderful classic work on tumors), still I abandon further attempts in this direction, and follow the anatomical principles in classification, passing gradually from tumors formed of simple tissues to those formed of more complicated tissues.

Lastly, I must mention that I voluntarily and intentionally limit my lectures to those cases of tumors which, in the commencement of the disease at least, are seated in parts of the body belonging to surgery. This limitation is not so important as it seems; we may even say that the peculiar course of tumors can only be studied in its purity, when they are located in parts where they do not directly endanger life; for the symptoms which they cause when in liver, stomach, or brain, are not those due to the tumors themselves, but are chiefly disturbances of function in the affected organ. If every typhus was accompanied by fatal intestinal hæmorrhage or perforation of the intestine, we should never have a pure representation of the disease proper, as its course would always be disturbed. We shall here and there remark on the relative frequency of primary localization of tumors in the internal organs, but cannot go into the symptomatology and histology of the diseased organ. On these points you will be instructed by the pathological anatomists and in the medical clinic.

[It has already been mentioned (page 606) that portions of foreign tissue may heal in the body of a living animal and be nourished there; and even more important for our knowledge about tumors is the fact that a tissue thus grafted may retain to some extent its peculiar physiological development: it is possible to introduce small fragments of periosteum from young animals into the lungs of living animals, where they may produce true bone; the same thing occurs when the transplantation is into the subcutaneous tissue. The periosteal fragments do not develop into a tumor, and the newly-formed bony plate is finally reabsorbed, but the experiment shows that the tissue-cells, which render the periosteum a bone-forming substance, may preserve their vitality independently and for a long time, even when nourished from strange vessels.

Besides spreading of tumors by cellular elements, it has been supposed that they are also conveyed by a contagion not connected with cells, but by the fluids of the neoplasm, which are said to have a purely chemical influence. It must be acknowledged that some cases placed in this class are difficult to explain in any other way. Thus malignant tumors with metastases to internal organs have been observed in pregnant women, while the fœtus presented a neoplasia of similar structure. Since at present it is generally supposed that solid elements do not pass from the maternal organs to the fetal placenta, these observations must await an exhaustive explanation.

Various observers have suggested, as causes for certain tumors, disturbances in the original germ-layers of the embryo, and lately *Cohnheim* has proposed a theory of the same kind. But it is not yet explained what this defect is: he thinks it simplest to suppose that in an early stage of embryonal development more cells are produced than are required, so that some remain unused; these may be small in amount, but from their embryonal nature have a great faculty for increasing. The time of this production of superfluous cells would be in a very early period—according to *Cohnheim*, between the complete differentiation of the germ-layers and the formation of special organs. It may further be supposed that the cell-material is either regularly spread over one of the histogenetic germ-layers, or remains at one point. "The latter would involve subsequently, local disposition to formation of a tumor; the former the beginning of a system, as of the skeleton or the skin." *Cohnheim's* hypothesis may explain many points in the development of tumors; he rejects entirely the etiological influence of external irritation, and recognizes error in embryonal disposition as the sole cause of neoplasia. This seems to me to be going too far; I think there is no doubt that local irritations may induce tumors, although the attempt to induce tumors in animals by injuries has not yet succeeded; but this proves just as little as the negative results of inoculating tumors. Moreover, we represent traumatic irritations too coarsely; they certainly had existed in cases where, as has often happened, tumors form at places long irritated by a fontanel or a wart, etc. To illustrate from another class, shall we not refer ulcers of the leg to local irritation, because we cannot induce them in animals by irritating their legs?

The question now arises, How does infection occur, and what are the means of locomotion of the specific elements of the tumors? The first means to look at are the blood and lymph vessels. For instance, if a tumor has opened a large vein, so that it can project into it, if the circulation be continued in the vein, particles of the tumor may be washed off and carried to various organs as emboli; but this is relatively rare; extension occurs much more frequently by means of the lymphatics. There is probably no direct passage of the cellular elements; at least, this has not been demonstrated. But it seems as if the walls of the vessels became diseased, while the caliber was obstructed by the tumor, and there was a direct propagation of the degeneration to the next lymphatic glands. That this mode is at least frequent is shown by microscopic examination of the cords, often perceptible on palpation, which unite the primary tumors with neighboring glands; it has not yet been shown how the tumor elements pass from the gland into the blood. Like the lymphatics, the blood-vessels also often show a progress of the disease, and this probably is the explanation of the occurrence of multiple disseminated tumors about the primary one. Quite lately *Axel Key* has stated that investigations by himself and *Retzius* show that the whole nervous system, peripheral as well as central, is provided with special serous channels communicating with each other, which may carry morbid germs in any direction. *Key* thinks that cells may be transported by these channels, and that this may account for the multiplicity of some tumors and the metastasis of others. This view is not yet sufficiently investigated, but seems not improbable.]

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## LECTURE XLVI.

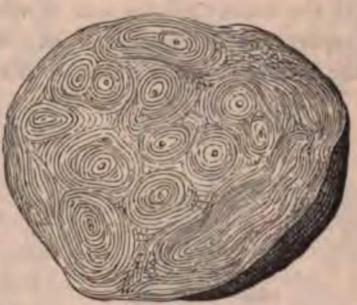
1. *Fibromata: a*, Soft; *b*, Hard Fibroma. Mode of Occurrence; Operations; Ligature; Écrasement; Galvano-caustic.—2. *Lipomata:* Anatomy; Occurrence; Course.—3. *Chondromata:* Occurrence; Operation.—4. *Osteomata:* Forms; Operation.

### 1. FIBROMA—FIBROUS TUMOR—CONNECTIVE-TISSUE TUMOR.

Tumors composed chiefly of developed connective tissue are called fibromata. They occur in the following forms: *a. Soft fibrous or connective-tissue tumors.* These are quite frequent, and are located almost exclusively in the cutis; they are composed of a very tough, somewhat œdematous, white tissue, and are usually covered by the thin papillary layer of the cutis. Microscopic examination shows loose connective tissue, as in the cutis. On the surface of the tumor there are almost always pointed papillæ, even when the tumor is developed in a part of the skin which normally has no papillæ; in the rete Malpighii of these formations, there is often a brownish pigment, which rarely extends deeper in the tissue; they may also have large vessels and abnormal enlargements of the hair and sweat glands on their surface; they are usually loosely hanging (cutis pendula, molluscum fibrosum), often distinctly pedunculated tumors; they might be termed partial hyperplasias of the skin, as they consist essentially of the elements of the skin. The growth is very slow, free from pain, and often goes on to the development of enormous tumors. Occasionally such growths are congenital; they may be multiple; hundreds of them may occur on the surface of the body. The congenital cutis-proliferation is most frequent on the face, generally unilateral, diffuse or in the shape of soft, cock's-comb-like vegetations. Freckles, pigmented hairy mother's-marks (moles, benignant melanoses, melanoma, pigmented fibroma) belong to this class. These tumors are apt to occur toward the end of middle life; in women, we not unfrequently find them hanging from the labia majora; as growths on this part are concealed as long as possible, they are usually quite large when first seen by the surgeon. *Virchow* terms the disease, in which these multiple, soft, fibrous tumors develop, *leontiasis;* in the course of time they are occasionally accompanied by general disturbances of nutrition. Although these tumors are not infectious, in the meaning we have attributed to this word, they occasionally lead to a cachectic state, and in the course of years to death by marasmus. There is also a relationship between this disease and Oriental elephantiasis, although by this name we mean a more nodular, but at the same time rather diffuse hypertrophy of the cutis of certain parts of the body (labia pudenda, scrotum, legs), which runs its course with repeated erysipelas. There would be less misunderstanding if these developments were briefly termed hypertrophy of the skin or pachydermata. Elephantiasis Græcorum is a similar disease as far as regards the cutaneous thickening, but it is strongly endemic, and is accompanied by some nervous symptoms; it occurs in Greece, Asia, and Norway (under the name of Spedalsked), and, after inducing long suffering, usually proves fatal.

*b. Firm fibromata, fibroid, desmoid tumors* appear to the naked eye to be composed of very firm, closely-interlaced fibrous tissue. They are always very hard, and of roundish or tuberous form; their cut surface is pure white, or pale reddish; to the naked eye many of them show on their cut surface a very peculiar, regular layering, and a concentric arrangement of filaments around distinct axes (see Fig. 115); according to my investigations, this results from the fibrous formation taking place around nerves and vessels, the latter being consequently embedded in the midst of the fibrous layers; frequently the nerves are thus destroyed.

FIG 115.



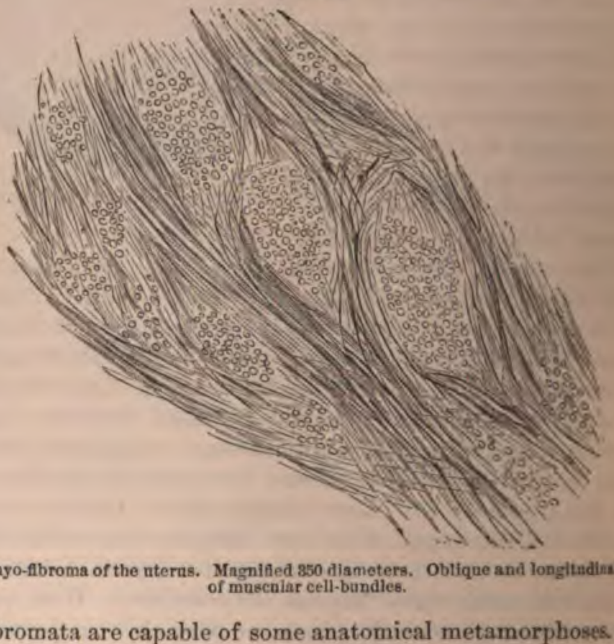
Small fibroma (myo-fibroma) of the uterus: natural size of the section.

With the external peculiarities just described, the histological appearance renders it difficult to classify these tumors. There can be no doubt that those of them which consist chiefly of connective tissue, such as old uterine fibroids, should be called fibromata; but the younger tumors of this variety, with the same appearance and consistence, show little connective-tissue but numerous spindle-shaped cells. The significance of these cells is varied. *Virchow* considers them muscle-cells; hence, what have hitherto been called fibroids of the uterus, he does not class among the fibromata, but among myomata, and terms them "myoma lævicellulare." If we consider fibre-cells as young connective-tissue, we must christen these tumors spindle-celled sarcoma or fibro-sarcoma. You see here, in apparently simple fibrous tissues, we become involved in difficulties with histology and histogeny.

[One reason for distinguishing this tumor from sarcomata is that in the uterus rapidly-growing recurrent tumors develop, in whose older parts the structure of fibroma predominates, while the younger portions contain quantities of spindle-cells—true sarcoma-cells. Clinically these tumors do not act like the much more frequent myomata, but like sarcomata. Some pathologists assume that in these cases there has been a development of a sarcoma in a fibroma; but it is more natural, from the predominance of the signs, to class these tumors as sarcomatous.]

There are two things that would induce me to regard fibro-cellular tumors as myomata: i. e., the oval and finally rod-like, wavy form of the nuclei, and the very distinct arrangement of the fibrous layers into bundles, while the individual fibre-cells are isolated with difficulty, perhaps only by aid of the recognized chemical means. At the same time the soil in which the tumor is developed is very important; the probabilities for a myoma would be very great if the neoplasia occur in the substance of the uterus.

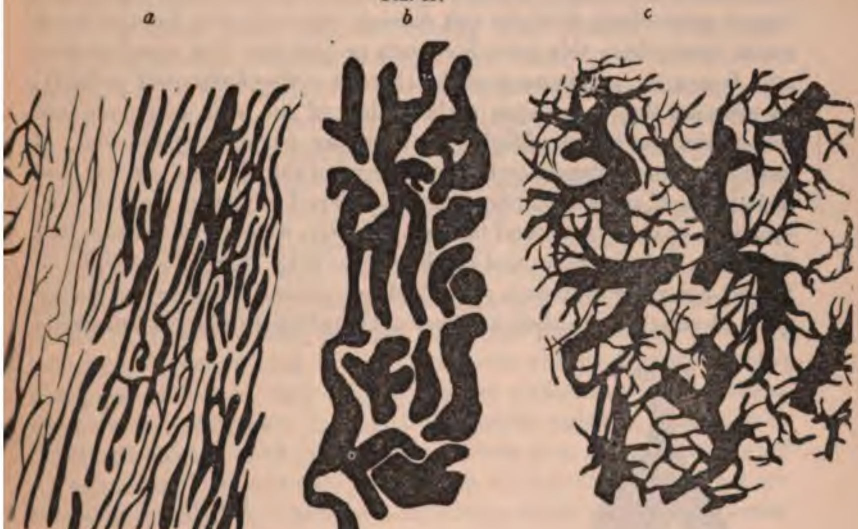
FIG. 116.



From a myo-fibroma of the uterus. Magnified 350 diameters. Oblique and longitudinal section of muscular cell-bundles.

Fibromata are capable of some anatomical metamorphoses. Partial mucous softening, great serous infiltration (brawny appearance and consistence), calcification, and even true ossification, are not very rare. Superficial ulceration is quite frequent in fibromata lying close under a mucous membrane; it results from external injuries in the usual way. The ulcer, thus formed, often shows good granulations and suppuration, and, under favorable circumstances, it may be brought to cicatrize. Fibrous tissue, though apparently poor in vessels, often contains quite a number, both of arteries and veins, as may be shown by injections; occasionally a very coarse cavernous net-work of veins forms in it (see Fig. 117); arteries and veins are so intimately united with the tissue of the tumor, that their adventitia mostly disappears in it, so that, in case they are injured, they cannot retract either transversely or longitudinally, and they remain gaping. This is the anatomical mechanical cause for bleeding from fibromata being so profuse, and why frequently it is not arrested without artificial aid. The rigid gaping opening of the vessel renders the formation of a thrombus very difficult. Occasionally, in large uterine and in periosteal fibromata, we find lacunar fissures filled with thin serum; possibly these are ectatic pathological newly-formed lymph sinuses; there are no certain observations on this point. Cavities, as large as the head, filled with serum, also occur in uterine fibromata (*Spencer Wells*).

FIG. 117

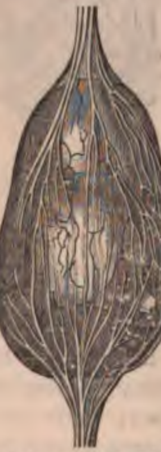


*a* and *b*, vessels of a cutis fibroma (myoma ?) from the thigh, injected through an artery; *b*, cavernous veins; *c*, peculiar regularly-arranged veins of a cutis-fibroma (myo-fibroma ?) of the abdominal walls, injected through a vein. Magnified 60 diameters.

The localization of fibroma varies greatly; of all the organs the uterus is most frequently affected (if under the general term "fibroid" we include myo-fibroma); here these tumors occasionally attain an enormous size, and then not unfrequently calcify. They are usually roundish, and are distinctly and sharply bounded: they are most frequent in the body of the organ, rarer in the neck, and hardly ever occur in the vaginal portion; their growth progresses upward and downward, that is, into the abdomen, gradually stretching the peritonæum, or through the os uteri into the vagina. In the latter direction, the tumors continue to grow, become pedunculated, and often give rise to severe hæmorrhages; they are called *fibrous uterine polypi.*

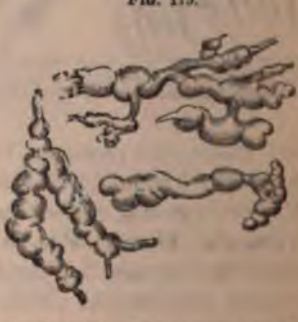
Fibromata, starting from the periosteum, are quite frequent; they are almost always fibro-sarcomata, i. e., they are composed of fibres and spindle-shaped cells, the latter may even preponderate (fibrous sarcoma, *Rokitansky*). The periosteum of the bones of the skull and face is particularly liable to this disease, especially the inferior turbinated bone; from this point fibromata project into the nasal cavities and fauces as polypous growths (fibrous *naso-pharyngeal polypi*); by pressure they may cause reabsorption of the bone and grow into the cranium or antrum Highmori; they are particularly vascular. I have also seen fibromata on the periosteum of the tibia and clavicle, and in bone itself, as in the upper maxilla, where I have met strange combinations of chondroma and fibroma. Lastly, we have to mention that fibromata are not rare in and on the nerves (Fig. 118). Frequently all tumors occurring on nerves are called *neuromata*, but they are distinguished according to their anatomical characteristics; most neuromata

FIG. 118.



Neuroma, after *Follin*

FIG. 119.



Small nodular fibro-sarcomatous neuromata from the eyelid of a boy; natural size.

are fibromata or fibro-sarcomata in the nerve-trunks; others consist partly or entirely of newly-formed nerve-filaments (*true neuromata*

Sometimes the nerve-fibromata follow the nerve-trunks and form nodular cords (plexiform neuromata, *Verneuil*) (Fig. 118), on whose confluence, as already stated, the peculiar appearance of the cut surface of the fibroma (Fig. 115) occasionally depends. Fibroma is rare in the subcutaneous cellular tissue; in the glands, except, perhaps, in the mamma, it hardly ever occurs.

The fibrous tumors just enumerated are particularly apt to develop in middle age (from thirty to fifty years); they are rarer in youth, and still more rare in advanced age. When we find them in the uterus of old women, there will probably have been there many years. Only fibroid neuromata, and bone and periosteal fibromata, occur in young persons, not exactly in children (though I saw one case of neuro-fibroma in a boy seven years old), but usually after puberty. Fibromata are somewhat more frequent in women than in men; uterine fibromata develop about the thirty-fifth to the forty-fifth year, although the trouble from them is often experienced later; they are rather more frequent multiple than solitary; periosteal fibromata usually remain solitary, but not unfrequently return, though, perhaps, not for years (regional recurrence; relation to sarcoma). Usually the growth of fibroma is purely central, and they are not infectious; but infectious fibromata are said to occur. Several such tumors near together unite, infiltrate the surrounding parts, and occasionally cause fibroid degeneration of the neighboring muscles, bones, and lymphatic glands. The infectious fibromata that I have seen were always fibro-sarcomata; like pure sarcomata, they may appear as metastases in the lungs. Fibromatous neuromata are quite frequently multiple, especially in different branches of the same nerve. Some time since I extirpated six neuromata from one man; three from the left arm, three from the left lower extremity. Cases have been seen where there were twenty or thirty neuromata at once.

Pure fibromata usually grow very slowly, and in age their growth is occasionally checked. This is best known of fibroma of the uterus, which usually ceases to grow after the change of life, and then often becomes calcareous. Combinations with other tissue-formations, especially with sarcoma, as already stated, occur, and take place in such a way that the primary tumors present a fibrous consistence, while the recurring tumors and secondary tumors resulting from infection are soft cellular sarcomata. I have seen such cases. A man about twenty-five years old, of healthy appearance, had a fibro-sarcoma as large as a walnut, in the abdominal walls; it was entirely removed; a new tumor appeared in the wound; subsequently several soft tumors appeared at other points on the surface of the body; at the same time the patient became marasmic and died in a few months; the whole lung was filled with soft sarcomatous tumors.

[Fibromata of the abdominal walls, which usually start from the sheath of the recti muscles or the aponeuroses of the oblique muscles and passing inward unite with the peritonæum, grow rapidly and in this respect resemble sarcoma. Lately I saw a woman, thirty-nine years old, who for years had a uterine fibroid as large as a child's head, in whom was developed a fibroma, the size of a plum, in the abdominal walls, which was painful spontaneously as well as on pressure. Possibly this was a neurofibroma.

Large, deeply-seated fibromata, with broad bases not accessible for operation, are met with. Surgery has made great advances in such cases of late years; and immense fibromata have been removed alone or with the uterus by abdominal section. Fibroids of smaller size, projecting into the uterus, after dilatation of the os uteri are enucleated and removed through the vagina. A reason for this operation not being done more frequently than it has been, is the slight amount of annoyance usually caused by such tumors; and the fact that in time they cease to grow, so that often it is unjustifiable to submit the patient to any dangerous operation. Of late, too, in large uterine fibroids subcutaneous injections of ergotine have proved successful. Under this treatment the tumors shrink away, or at least the hæmorrhages cease.]

After what has been said, the diagnosis of fibroma is not difficult, the consistence, locality, age, mode of attachment, and form of the tumor, almost always lead to its correct recognition.

The *treatment* consists exclusively in the removal of the tumor. When practicable, this is generally done with the knife; but pedunculated or hanging connective-tissue tumors and fibrous polypi admit of other methods of operation. Formerly the ligature was much resorted to in such cases, i. e., the pedicle of the tumor was tied tightly with a thread, so that it became gangrenous and fell off; this method was chosen especially in cases where bleeding from the cut surface was feared. Ligation has the great disadvantage that then the tumor decomposes in or on the body, and that the ligature must be tightened several times before it cuts through; this may induce severe hæmorrhage. The ligature may be combined with incision, by cutting off the tumor in front of the ligature, and leaving only part of the pedicle to become detached spontaneously. In the nares and pharynx, as well as in the vagina, there is of course great difficulty in applying a ligature, and for this purpose numerous instruments, simple and complicated, so-called loop-bearers, have been constructed, by means of which the ligature is passed over the tumor on to the pedicle. But the ligature is now so generally rejected and so little used, that all these instruments, some of which are very ingenious, are for the most part only of historical value.

[Simple ligation of tumors is rarely done now; in its place is used the elastic ligature, which is more energetic and certain. For this purpose simple rubber drainage-tubes of various caliber, or solid rubber ligatures, are employed. The whole pedicle may be ligated at once or in portions, by thrusting a trocar through the base of the pedicle and passing ligatures through the canula, and ligating different portions. The elastic ligature should be drawn as tight as possible, so as to cut off the circulation; to prevent the ligature from slipping, two needles may be thrust through at right angles between the ligature and tumor. When the ligation has been done, the ligatures cut through quite soon; but this is not generally waited for, the tumor being often removed by the thermocautery; in doing this it is well to keep below the needles, so the ligature may hold well.]

But the desire to remove pedunculated tumors without hæmorrhage is still strong, and has lately led to new instruments and new methods, which, however, could not have become popular before the introduction of chloroform. *Crushing* and *burning* off have now taken the place of the ligature. *Écrasement* as done by *Chassaignac* we have already described; this operation, if done slowly, is followed by no hæmorrhage, even from arteries of the diameter of the radial; the resulting wound is perfectly smooth and regular, and heals well without much sloughing from the surface; although hæmorrhage is not certainly avoided in all cases, it is in most; the instrument is made of various sizes; the smallest may be passed into the nose, and with it we may readily crush off small pedunculated naso-pharyngeal polypi. The *galvano-caustic* of *Middledorpf* is a method of similar effect; its object is to heat a loop of platinum wire between the two poles of a galvanic battery, and with it burn through the base of the tumor; the result is a simultaneous division and arrest of hæmorrhage; the latter fails about as often as it does in écrasement, that is, very rarely—hence this method is advisable in certain cases. The trouble in preparing a strong, active battery (which is quite expensive) is such that galvano-caustic will probably never come into general use; in spite of its elegance, it has been strangled almost at its birth by the introduction of the écraseur; the medical public has already decided the question; almost every operating surgeon has an écraseur, only a few hospitals have galvano-caustic apparatuses.

As regards operation for non-pedunculated, more deeply-seated fibromata, some of them are not at all accessible to surgical treatment; we cannot recommend cutting uterine fibromata out of the abdomen, not because the operation is excessively dangerous, but because, in the course of time, these tumors usually come to a stand-still, and the annoyance they cause rarely balances the danger to life. As regards those fibromata, also, which are not dangerous from their seat or growth, but to operate on which would be dangerous, we should bear in mind that these tumors grow very slowly, often come to a halt in advanced life: hence we should not undertake such operations too hastily, or urge them too strongly. But there are many cases where we may and must operate without hesitation; extensive, frequently-repeated hæmorrhages from an ulcerated fibroma, threatened destruction of bone, or protrusion into the skull, are urgent indications. In neuro-fibromata the pain is sometimes so severe that the patients strongly urge operation, even if we have to tell them that paralysis of the parts supplied by the nerve affected would be the necessary result, for we almost always have to excise a portion of the diseased nerve which possibly still performs part of its functions. If the neuroma be painless, it would be foolish to excise it.

### 2. LIPOMATA—FATTY TUMORS.

Of course, the disposition to formation of fat, when it does not exceed a certain point, is not regarded as a morbid diathesis, but rather as a sign of good nutritive condition, and varies with the age, being greatest between the thirtieth and fiftieth year, and being essentially favored by a quiet, pleasant life and phlegmatic disposition. We only begin to regard it as a disease when it induces functional disturbance of different organs, or of the organism at large, or if the development of fat be limited to a small part of the body, when it appears as a fatty tumor.

[Fatty tumors, or lipomata, are analogous to the normal type of fatty tissue in the embryo: *Toldt* has found that in the embryo the fatty tissue, which subsequently is diffused, develops at certain parts of the body as distinct lobular masses, characterized by a supply of blood independent of the neighboring tissues. The peculiar cells, which *Toldt* considers distinct from connective tissue, during intrauterine life contain usually no large drops of fat; hence to the naked eye they closely resemble gland-tissue.]

The anatomical formation of fatty tumors is simple; they consist of fatty tissue, which, like the subcutaneous fat, is divided into lobes by connective tissue. This connective tissue may be more or less developed, and the tumor may consequently be sometimes firm (fibromatous lipoma), sometimes softer (simple lipoma). The shape is usually round and lobular, and the fatty mass separated from the adjacent structures by a thickened layer of connective tissue (circumscribed lipoma, the usual form), and may readily be separated from the parts around; more rarely, lipoma appears as a corpulence limited to one part of the body, as a swelling without distinct boundaries (diffuse lipoma). The seat of lipoma is most frequently in the subcutaneous cellular tissue, especially of the trunk; these tumors are most frequent on the back and abdominal walls; they are rarer on the extremities; in the synovial folds and tufts of the joints, as well as in the sheaths of the tendons, there may be an abnormal development of fat, so that the fatty masses may seem branched like a tree (lipoma arborescens, *J. Müller*); this is an analogy to the fatty proliferation in the processes of the peritonæum of the colon (appendices epiploicæ) and other serous membranes, but it is exceedingly rare. The growth of lipoma is always very slow, its development is hardly ever accompanied by pain, unless it comes close to a nerve and presses on it, which rarely happens. Fatty tumors may attain a great size; the patients, being little troubled by them, rarely feel obliged to have them removed early. Hence lipomata grow to enormous tumors; recently I removed one from the back of a woman; it began under the right scapula and reached down to the calves; above, at its base, it was the same circumference as the larger part of the patient's thigh, below it was almost twice as large. Secondary changes in these tumors are not very frequent, but the thick connective-tissue partitions in the tumor may calcify, or even ossify, and at the same time the fatty tissue may change to an oily or emulsion-like fluid. The skin covering the tumor is gradually expanded, and at first is usually much thickened, and occasionally colored brown, but generally remains movable over the tumor; exceptionally there is an intimate adhesion with the newly-formed fat, and then a superficial ulceration of the cutis, which in such cases is entirely atrophied; this ulceration, which may be induced by external irritation, rarely goes deep, although parts of the fatty tissue may become gangrenous; under such circumstances there are almost always formed ulcers with slightly-developed granulations and serous, badly-smelling secretions. Combinations of lipoma with soft fibroma, with myxomatous sarcoma, and with lymphoma, do occur, although rarely. In lipoma I have several times seen considerable cavernous dilatation of the veins.

A *disposition* to the development of lipoma most frequently exists at the time of life when the tendency to development of fat generally is greatest, between the thirtieth and fiftieth years; in children it is very rare, still it occurs congenitally on the back, neck, face, as well as on the toes, with coincident hypertrophy of the bones (giant growth); they grow little after birth. Usually there is only one lipoma, and it grows very slowly; indeed, it may remain at one point, especially in old persons. In the subcutaneous cellular tissue, development of multiple lipoma has been frequently seen; cases have been noted where fifty or more, usually small lipomata, were developed at once; subsequently they ceased to grow. Multiple lipomata are often mixed tumors. Simple lipoma is *never* infectious; hence it never recurs after extirpation.

Pressure and friction are occasionally observed as exciting causes for the development of fatty tumors; there is also a moderate degree of hereditary influence in fatty disease generally.

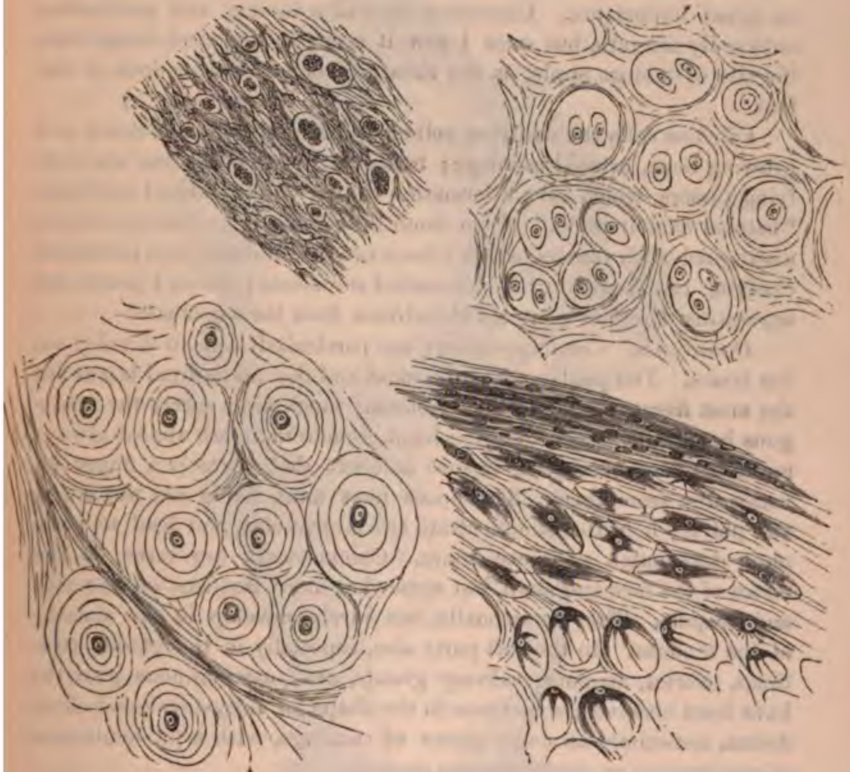
The diagnosis of lipoma is generally easy; the consistence, the lobular feel, occasionally a perceptible crackling, from compression of individual fat-lobules, are the objective symptoms; other aids for confirming the diagnosis are, the movability of the tumor, the slow growth, age of the patient, and, above all, the region of the body; there is a possibility of mistaking them for fibrous tumors, sarcomata, lipomatous-cavernous blood-tumors.

The *treatment* consists in removal with the knife. Healing is usually preceded by free discharge of gangrenous tissue from the wound; in very large lipomata it is best always to remove a portion of the skin covering it, with the tumor; after their extirpation erysipelas is quite frequent, especially in very fat patients. The largest lipomata may be removed with good result, as they usually occur in persons otherwise healthy. Extirpation of diffuse lipomata is more unfavorable than that of the circumscribed; the local and general reaction is usually more considerable, but I have several times performed such operations with good results.

### 3. CHONDROMATA—CARTILAGE-TUMORS.

These are tumors consisting of cartilage, of the hyaline or fibrous variety. The *microscopic elements* of pathological, newly-developed cartilage may vary; occasionally we see exceedingly beautiful round cartilage-cells, such as are particularly found in the embryo, and somewhat smaller in the articular and costal cartilage; but such a complete change of hyaline substance to a homogeneous mass, as is the rule in normal cartilage, is more rare in chondromata; frequently the intercellular substance pertaining to the different groups of cells is distinct, and between the large groups of cells the hyaline substance forms fine filaments. The latter is the cause of sections of cartilage-tumors having the appearance of being traversed by capsular-like, communicating connective-tissue meshes, which even to the naked eye show a kind of net-work; the bluish or yellowish glistening cartilage is seen embedded between these connective-tissue striæ. The tissue of chondroma also distinguishes itself from that of normal cartilage by the fact that the former is usually vascular in the above-mentioned fibrous striæ, while, as is well known, the latter has no vessels. The microscopic appearances in chondroma have still some other points of difference from those of normal cartilage. Not unfrequently the intercellular substance, whether hyaline or slightly striated, instead of having the regular firm consistence of normal cartilage, is more gelatinous or friable, or possibly becomes so secondarily. Calcification of the cartilage, as well as true ossification, is quite frequent in chondroma; the forms of the cells may vary greatly (Fig. 120).

Fig. 120.



Extraordinary forms of cartilage-tissue from chondromata, taken from men and dogs. Magnified 350 diameters.

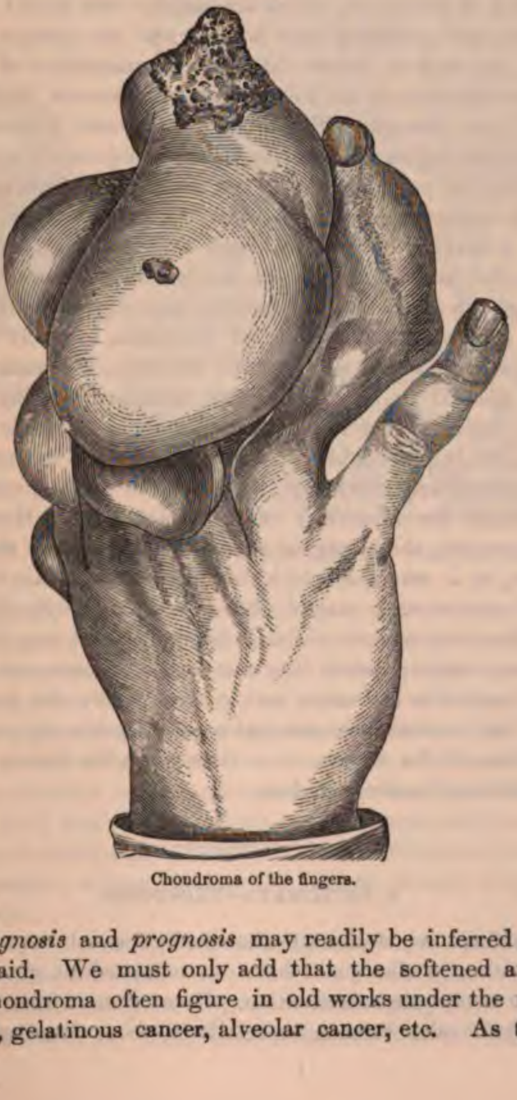
In shape, chondromata are usually roundish, nodular, sharply-bounded tumors, which may grow to the size of a man's head, or larger. At first their growth is almost purely central; subsequently, however, the tumor enlarges, partly from the occurrence of new foci of disease in the immediate vicinity, partly from transformation of the adjacent tissue into cartilage (local infection). Among the anatomical metamorphoses, the pulpy and mucous softening, and the ossification of individual parts, have been already mentioned; the former causes mucous cysts in these tumors, which give a feeling of partial fluctuation to the otherwise hard chondroma. It is imaginable that, with complete ossification of the chondroma, the tumor would cease to grow; and this has been seen in some cases, although rarely. In large chondromata superficial ulceration is apt to occur, especially if the skin is very tense, or from occasional traumatic irritation, but it is of no great importance. Ulcerative central softening and perforation outwardly are rare, but once I saw it occur in a typical chondroma, the size of a large apple, on the sheath of one of the tendons of the foot.

*Virchow* calls the ossifying cell-layer between the periosteum and growing bone, osteoid cartilage; hence he terms periosteal and ossifying tumors, which have a formation similar to this osteoid cartilage, "osteoid chondromata." I am doubtful about any one being able to distinguish such tumors, which I have often examined, from periosteal ossifying round-celled or spindle-celled sarcomata; hence I prefer not separating *Virchow's* osteoid chondroma from the sarcomata.

*Occurrence.* Cartilage-tumors are particularly apt to develop on the bones. The phalanges of the hand and the metacarpal bones are the most frequent seat of chondromata; much more rarely the analogous bones of the foot. On the hand, chondromata are almost always multiple; they even occur in such numbers that scarcely a finger remains free from them. The bones next most liable are the femur and pelvis; here the tumors attain the largest size, and lead to complete destruction of these bones. Chondromata are rarer on the bones of the face and skull, but somewhat more frequent on the ribs and scapula. They occasionally, but rarely, develop in the sheaths of the tendons. In the soft parts also, especially in the glands (testicles, ovaries, mammæ, salivary glands, etc.), cartilaginous growths have been observed, sometimes in the shape of fully-developed chondroma, sometimes as single pieces of cartilage, with a predominance of sarcomatous or carcinomatous growth.

The development of chondroma is chiefly peculiar to youth; not that it occurs exactly in children, but shortly before the age of puberty. Most chondromata are referable to this age, even if they are first recognized much later in life. The tumors occasionally develop after injury, grow very slowly for twenty or thirty years, and occasionally seem to cease growing entirely. I have heard patients assert that the tumors had remained unchanged for years, and some accidental cause made them desirous of having them removed. Sometimes they grow more rapidly and become infectious; cases are known where cartilaginous tumors have appeared even in the lungs (embolic) and caused death. *O. Weber* has also observed an hereditary chondromal diathesis. In the combinations of cartilage-formations with sarcoma or carcinoma, the former has no effect on the prognosis of the tumor as a whole.

Fig. 121.



Chondroma of the fingers.

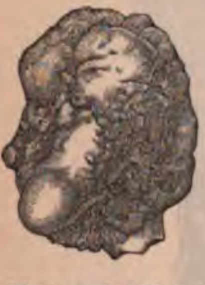
The *diagnosis* and *prognosis* may readily be inferred from what has been said. We must only add that the softened and cystoid forms of chondroma often figure in old works under the names colloid tumors, gelatinous cancer, alveolar cancer, etc. As the epithelial elements and connective-tissue framework may become gelatinous (mucous, colloid, myxomatous) in fibroma, chondroma, and sarcoma, as well as in adenoma and glandular cancer, we must always observe very particularly what we have before us: frequently we shall be in doubt about the significance of the histological elements, as well as about the proper name.

The only *treatment* is removal of the tumor, if it can be done without endangering life. Of course we would not interfere with the chondromata of the pelvis, which are usually very large; those of the thigh, which are generally very large when the patient applies for treatment, can only be gotten rid of by exarticulation of the femur, and we should scarcely do this before spontaneous fracture of the extremity, from disease of the bone, has rendered it useless. Chondromata of the fingers are most frequently subjects for operation, not because they are painful, for they are usually free from pain, but because they impair the function; this takes place very slowly and gradually, hence the tumors will have attained a considerable size. So long as the patients can use their nodulated swollen fingers, they neither urge the operation, nor can we urgently advise them to submit to it. As regards the mode of operation, in many cases where the tumor, even if firmly adherent to the bone, is seated laterally, it would be natural to try dividing the skin, and pushing it and the tendons to one side, then removing the tumor with the knife or saw. But this is rarely practicable, if we would remove the entire tumor, which is imperatively necessary; for often the cartilaginous mass entirely pervades the medullary cavity of the bone. Moreover, after such an operation, there may be severe inflammation of the sheath of the tendon, as a result of which the finger may remain stiff. There have not been enough careful observations to verify *Dieffenbach's* assertion, that any remnants of the chondroma that may be left ossify and become stable; hence the removal of chondroma from bone should be limited to few cases, and to those where the tumor is still small. If the tumors have attained a considerable size, we postpone exarticulation of the fingers to a time when the tumors shall have rendered the hand entirely useless.

## 4. OSTEOMATA—EXOSTOSES.

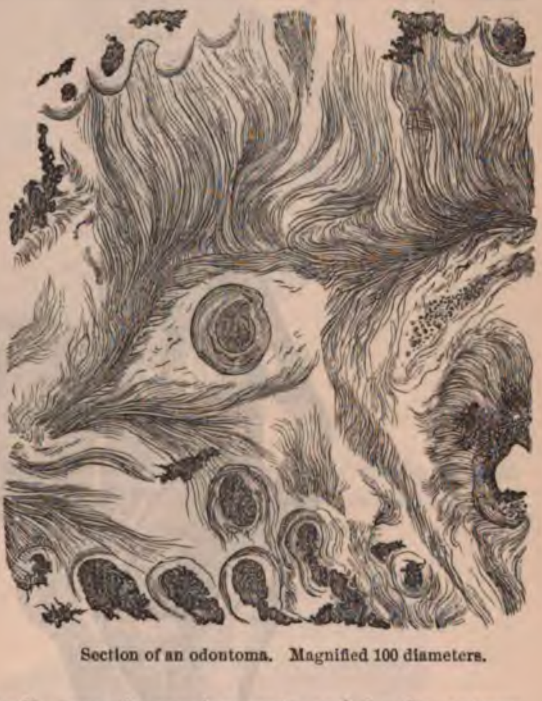
By this term we designate abnormally-formed masses of bone, which are circumscribed, and have an independent growth, not depending on a chronic inflammation. Formation of bone also occurs occasionally in other tumors, especially in those forming in bone, as we have already remarked when speaking of chondroma. But the name osteoma is usually limited to tumors consisting entirely of bone. I may mention here that not only new formations of entire teeth (very irregularly shaped) occur in ovarian cysts and in the antrum Highmori, but that on the teeth themselves outgrowths of true ivory matter, *ivory exostoses* (odontoma of *Virchow*) have been observed; but these are very rare, and may be regarded merely as curiosities. Exostoses consist partly of spongy bone-substance, like that in the medullary cavity of bones, partly of ivory-like substance, like that in the regular lamellæ of the cortical substance of the hollow bones; hence we shall distinguish *spongy exostoses* and *ivory exostoses*. A third form of osteomata is formed by the ossification of tendons, fasciæ, and muscles, whose right to be classed among tumors is, however, doubtful.

FIG. 122.



Odontoma of a back tooth, natural size.

FIG. 123.

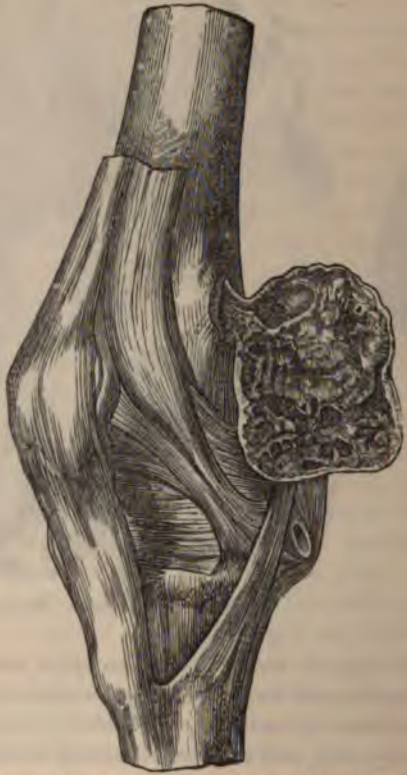


Section of an odontoma. Magnified 100 diameters.

(*a.*) *Spongy exostoses*, with cartilaginous covering (exostosis cartilaginæ). These tumors occur almost exclusively on the epiphyses of the long bones; they are outgrowths from the epiphyseal cartilages, whence *Virchow* very properly calls them "*Ecchondrosis ossificans*"

(Fig. 124). On their roundish, nodular surface, there is a layer of beautifully-developed hyaline cartilage, about a line or a line and a half thick, which evidently grows partly in itself, partly peripherally from the periosteum or perichondrium, then rapidly ossifies toward the centre. The newly-formed bony mass itself is, from its start, most intimately connected with the spongy substance of the epiphyses, so that the hard tumor is immovably seated on the bone. From the nature of these exostoses they can only occur in young persons. According to my observation, tibia, fibula, and humerus, are their most frequent seat.

FIG. 124.

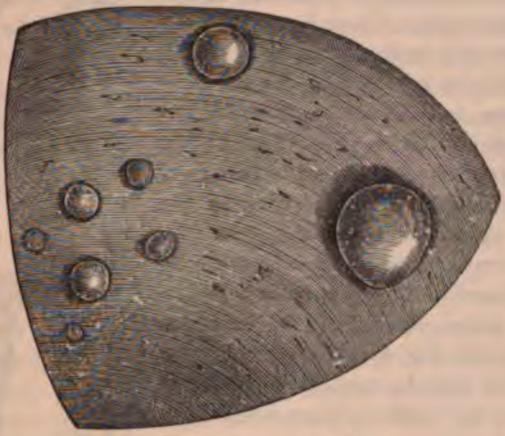


Pedunculated spongy exostosis from the lower end of the femur, after *Pean*.

(*b.*) *Ivory exostoses.* These consist of compact bony substance, with Haversian canals and lamellar systems; they develop on the bones of the face and skull (Figs. 125 and 126), on the pelvis, scapula, great toe, etc., and form roundish, nodulated, or smooth tumors.

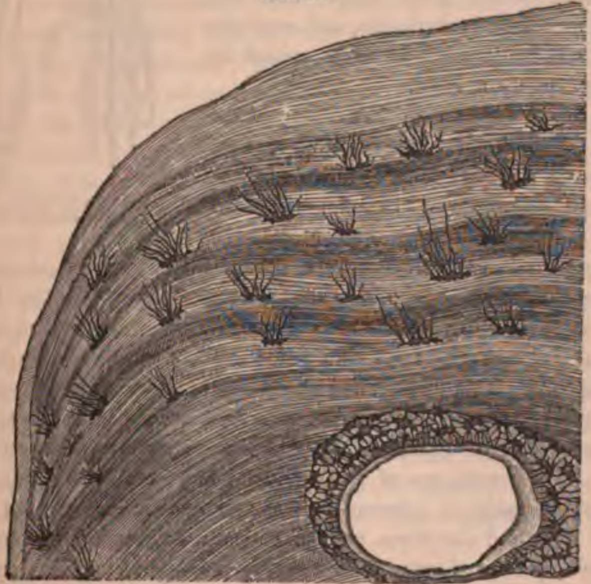
A third variety of tumor-like formation of bone is the abnormal ossification of tendons, fasciæ, and muscle, which usually occurs on a series of tendons and fasciæ after they have previously ossified a great deal, so that the skeleton of such patients, who are generally young,

FIG. 125.



Ivory exostosis of the skull.

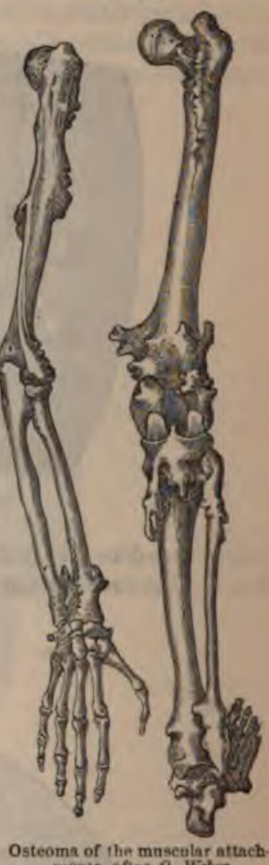
FIG. 126.



Section from an ivory osteoma of the skull.

are covered with twenty to fifty long, sharp, bony processes, where the tendons are attached to the bone (Fig. 127); as in one case observed in Zürich, the ossification occasionally occurs in the fascia of the muscle. Cases have been observed where this ossification was so extensive that all the muscles of the shoulder and arm were ossified, and the upper extremity could not be moved. These bony neoplasiæ, as well as the so-called *exercise-bones*, must doubtless be regarded as the product of chronic inflammation, just like the true bony formations that are abnormally developed in the membranes of the brain and spinal medulla. By exercise-bones we mean the development of bone in the deltoid muscle, particularly at those points where the musket strikes when drilling. But these bones form in few soldiers, and their development presupposes a tendency to the formation of bone. Ossification of the tendons, especially of their points of attachment to the bone, which occasionally occurs from some unknown cause, is also very remarkable, and reminds us of a similar process in birds, which in them is perfectly normal.

Fig. 127.



Osteoma of the muscular attachments, after *O. Weber*.

The predisposition to formation of osteomata is allied to that for development of chondromata; it also occurs more frequently in the young, and in men than in women, while children almost escape it. As regards epiphyseal osteomata, which might be termed ossifying chondromata, they of course cannot occur later than the twenty-fourth year. But other exostoses also occur generally before the thirtieth year; observations on this point are not very numerous, as the disease is rare. This experience about the occurrence of osteomata in the young is the more remarkable, as it stands in a certain contrast to the general rule of ossification being especially apt to occur in old persons. The cartilages of the ribs and larynx and the spinal ligaments often ossify in advanced age; the chalky deposits in the arteries of the aged also form part of the almost natural senile marasmus; development of osteomata, however, rarely occurs in old persons, but when such tumors are found in them they have usually developed in youth. Osteomata are just as often multiple as solitary; their growth is generally very slow, and is usually arrested with advancing age. The growth of epiphyseal exostoses ceases after the skeleton has completed its growth, and its spongy substance becomes more compact. Ossification of the tendons and muscles rarely goes so far as to entirely prevent motion. In some cases development of bone has been observed in the lung. The inconveniences caused by osteomata are not usually great; their development is not accompanied by pain, nor are they sensitive to the touch; but osteomata in the vicinity of joints often impair their function. When these tumors occur on the bones of the face, they cause unpleasant deformities; exostoses on the big-toe prevent wearing the shoe; ossification of the tendons and muscles impairs or entirely prevents motion; but unfortunately, from their size and number, operative surgery can do little for the latter, and the less so, as the tendency to morbid development of bone still continues. The operation for exostosis consists in sawing or chiselling the tumor from the bone affected. But, as the latter is occasionally in the vicinity of a joint, the articulation might thus be opened; it is neither advisable nor necessary to undertake such operations unless the impairment of function be so great as to balance an operation dangerous to the joint and to life. We should be the less inclined to undertake such operations without some special indication, as in the course of time these tumors cease to grow. On epiphyseal exostoses we occasionally find mucous bursæ containing adherent, or loose ossifying chondromata; these mucous bursæ usually communicate with the joint in whose vicinity the exostosis is situated. According to the investigations of *Rindfleisch*, the mucous bursæ are always abnormal elongations of the pockets of the articular synovial membrane. I once allowed myself to be induced, by the entreaties of a patient, to remove such an exostosis on the lower end of the femur with a large mucous bursa; the patient died of septicæmia. In another case the mucous bursa over an exostosis on the lower end of the humerus opened spontaneously after moderate inflammation; there was suppuration of the elbow-joint, with anchylosis; the patient would not permit resection of the joint.

[It would seem, from observations of English surgeons in China, that elephantiasis Arabum depends on the filaria sanguinis hominis, which enters the system by the drinking-water and probably takes up its position somewhere in the lymphatic system. Patients with elephantiasis Græcorum are also said to have a peculiar bacillus in the blood.]

## LECTURE XLVII.

5. Myoma.—6. Neuroma.—7. Angioma: *a*, Plexiform; *b*, Cavernous.—Operations.

### 5. MYOMATA.

AT present it remains undecided whether there are *pure myomata*, i. e., tumors consisting entirely of transversely-striated muscle-filaments or their cells; I do not know that any such have been observed. The occurrence of newly-formed transversely-striated muscle-filaments has been very rarely observed in tumors. No tumor was ever entirely composed of them; they were usually an accidental occurrence in sarcoma or carcinoma (of the testicle, ovary, or mamma), or in tumors of very complicated formation; according to the latest observation, they are most frequent in sarcomatous tumors of the kidneys in children. I have examined tumors in which there were distinct stages of development of muscular filaments, but the right of classing such tumors as myomata has been disputed. I can say little against this, as we cannot call tumors, consisting of grades of development of connective tissue, fibromata, and as I formerly objected (page 685) to terming uterine fibromata, composed of spindle-cells, myomata, as we are not quite sure of the relation of spindle-cells to muscle-cells. In old men, extensive newly-formed smooth muscles occur in the prostate, partly as independent nodules, partly as diffuse enlargements of the organ. There is certainly no objection to terming these so-called prostatic hypertrophies (there is usually some coincident glandular) myoma; similar myoma-nodules are met in the muscular coat of the œsophagus and stomach. I have often removed from the bladders of young persons true myomata, of flat muscle-filaments (myoma læviculare), sometimes with pedicles; they seem to arise from the muscular coat of the bladder. Clinically, nothing certain can be said of myomata in these conditions; the tumors which I considered as young myomata in the muscles had, on section, a medullary fascicular appearance, an insuperable tendency to local recurrence, and thus caused death.

### 6. NEUROMATA.

It has already been mentioned (page 688) that the name "neuroma" is often applied to tumors occurring on the nerves; this is, if you please, a practical misuse, which, however, it is difficult to root out. By "true neuroma" we mean a tumor composed entirely of nerve-filaments, especially of those with double contours; they appear to come only on nerves, and are very rare. Neuromata in amputation-stumps have already been mentioned (page 125); many doubt whether there are any other true neuromata. True neuromata are always very painful. Many of the fibromata on and in nerves contain very peculiar bundle-like fine filaments richly supplied with nuclei, which may very well be taken for gray filaments containing no medulla, as *Virchow* considers them; this would make true neuromata a large class, and divide them into myeline and amyeline forms. I do not always trust myself to distinguish an amyeline neuroma from a fibroma in a nerve, and hence should not require it of others. Tumors composed of spindle-cells arranged in bundles are probably far oftener young myomata and neuromata than young fibromata, but it would be difficult to prove to which class they belong. Multiplicity and tendency to regional recurrence are peculiar to neuromata, hence the prognosis should always be guarded. It is rarely possible to dissect a neuroma from the nerve; part of the latter must generally be removed with it.

[Diffuse thickenings of all the twigs of a nerve-plexus are not very rare, and are called plexiform neuromata; they are sometimes accompanied by elephantiasis-like thickening of the skin and subcutaneous tissue of the parts supplied by the affected nerve (*Czerney*). Although the greater part of the plexiform neuroma is newly-formed connective tissue, still, young nerve-filaments are found through it; sometimes some of the nodules of a plexiform neuroma have entirely the character of a rapidly-growing sarcoma, with metastases to internal organs. We may distinguish two types of neuromata, those that come multiple on cerebral and spinal nerves, somewhat resembling fibromata, and solitary neuroma often mistaken for sarcoma. While in the new neuromata we have described there were no nerve-cells, only nerve-filaments, in some tumors of the brain and medulla oblongata we find new ganglion-cells. Quite lately *Axel Key* observed the same in a tumor, the size of a plum, that originated from the infraorbital nerve. In the soft tissue whose gross features were those of a sarcoma, he found numerous elements resembling ganglion-cells; but this is rare.

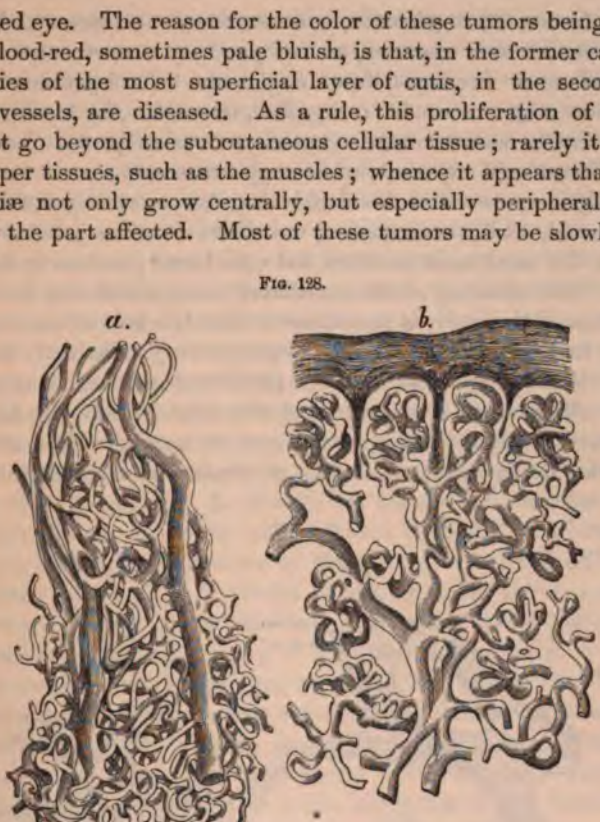
In neuroma the prognosis should be reserved; operations should only be done when the tumor grows rapidly or is very painful; if it is necessary to cut out part of the nerve, the ends should be sewed together if possible, or we may transplant portions of a nerve from some animal. This operation has succeeded experimentally; whether it would do so in man is a question worth deciding.]

### 7. ANGIOMATA—VASCULAR TUMORS.

By this term we mean tumors composed almost exclusively of vessels held together by a slight amount of connective tissue; they have also been called [nævi, mother's-marks] "erectile tumors," being firmer or softer, larger or smaller, according to the fulness of the vessels. The ordinary forms of varicose dilatations of the veins and the aneurisms of different arteries are excluded by this definition. But circoid aneurism and some forms of aneurismal varix might be classed here; yet, as this is not customary, we treated of these diseases earlier. Here we have to consider two different varieties of vascular tumors:

(*a.*) The *plexiform angioma* or *telangiectasis* (from τελος, αγγειον, ἐκτασις). This is the most frequent form; this neoplasia is composed entirely of dilated and tortuous capillaries, and anastomosing vessels, and, according as the proliferation of the vessels or the pure ectasia predominates, it appears more as a tumor or as a red spot on the skin. Plexiform angiomata, of the variety we are about to describe, occur almost exclusively in the cutis. They have sometimes a dark-cherry, at others a steel-blue color; are sometimes as large as a pin-head, again as large as a hemp-seed; some are moderately thick, others scarcely rise above the level of the skin. There are very rare forms where there is not a red spot or a tumor, but a diffuse redness over a large surface; in such cases, even with the naked eye, we usually see the distended and looped fine vessels on the surface of the cutis, showing through the epidermis. Anatomical examination of large extirpated angiomata of this variety shows that they are composed of small lobuli as large as a hemp-seed or a pea; and, if, after artificial injection or other mode of preparation, we examine them microscopically, we shall find that these lobuli are formed by the vessels of the sweat-glands, hair-follicles, fat-glands, and fat-lobuli, being independently diseased, and that the different small proliferating, vascular systems form the above-mentioned lobuli, which are visible to the naked eye. The reason for the color of these tumors being sometimes blood-red, sometimes pale bluish, is that, in the former case, the capillaries of the most superficial layer of cutis, in the second, the deeper vessels, are diseased. As a rule, this proliferation of vessels does not go beyond the subcutaneous cellular tissue; rarely it affects the deeper tissues, such as the muscles; whence it appears that these neoplasiæ not only grow centrally, but especially peripherally, and destroy the part affected. Most of these tumors may be slowly emptied by pressure, and again fill as soon as the pressure ceases. But there are also moderate-sized telangiectases, in which, besides the proliferation of vessels, there is also a new formation of connective tissue and fat, so that they cannot be entirely removed by pressure. When these new formations were superficial in the cutis, and the blood has been emptied from them after extirpation, with the naked eye we can hardly see any thing abnormal in the morbid piece of skin that has been removed; a moderate neoplasia of this variety appears on the cut surface as a pale-reddish, soft, lobulated substance, in which we can see no vessels with the naked eye, because the whole disease is usually limited to the capillaries and minute vessels, and to a few small arteries.

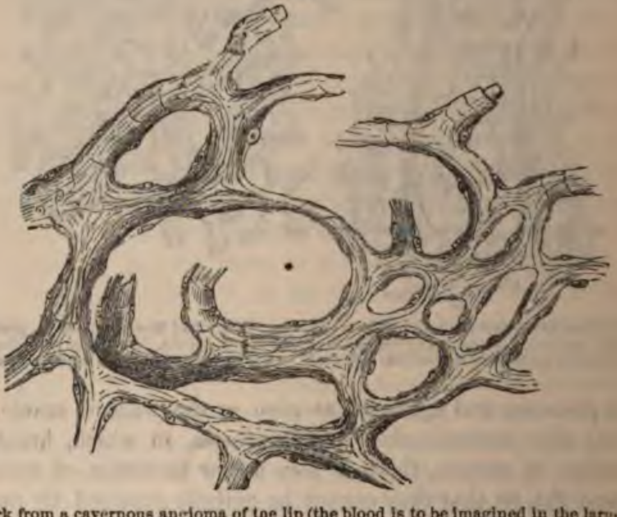
FIG. 128.



Conglomeration of vessels from a plexiform angioma. Magnified 60 diameters. *a*, proliferating vascular net-work around a sweat-gland (which is not shown, to prevent complicating the drawing); *b*, proliferating vascular net-work in the papillæ of the oral mucous membrane.

(*b.*) *Cavernous angiomata, or cavernous venous tumors.* We will first determine their anatomy, so that you may at once correctly note their difference from plexiform angiomata. Extirpated cavernous angiomata may at once be recognized, on section, by having almost exactly the formation of the corpus cavernosum penis. You see a white, firm, tough net-work, which appears empty, or at least contains only in spots red or discolored coagula, or possibly is filled with small, round, chalky concrements, so-called vein-stones; but we must imagine the mesh-work as distended with blood previous to its extirpation. The boundary of this cavernous tissue, which may form in all the tissues of the body, is sometimes evidently a sort of capsule; but in other cases this cavernous degeneration is very indistinctly bounded, and at different spots, in a rather indifferent manner, it enters the tissue. *Microscopic examination* of this mesh-work, which is formed sometimes of thin threads, sometimes of membrane-like capsules, shows that the branches are formed of remains of the tissue in which the cavernous ectasia occurs. The inner wall of the space filled with blood is, in most cases, coated with spindle-shaped cells (venous endothelium), so that even these anatomical conditions go to prove that we have to deal chiefly with distended veins. The mode of development of this peculiar tissue has received different explanations.

FIG. 129.



Mesh-work from a cavernous angioma of the lip (the blood is to be imagined in the large meshes between the net-work). Magnified 350 diameters.

If we had any accurate investigations about the development of the corpus cavernosum penis, we might draw some definite conclusions from them, on account of the great analogy of the two tissues. The three chief hypotheses about the development of cavernous tumors are as follows: 1. It is asserted that the cavernous spaces first develop from the connective-tissue, and secondarily become connected with the vessels; and it has even been suggested that blood might be developed outside of the circulation, from the derivatives of the connective-tissue cells; the striæ of the mesh-work would increase by independent growth, by sprouting, and club-shaped growth of the connective tissue (*Rokitansky*). This hypothesis, especially the formation of blood outside of the circulation, has some objections. 2. It is asserted that circumscribed dilatations of small veins occur close together, and that at the points where they come in contact the walls are gradually thinned or entirely disappear. This view is supported by the fact that these gradual distentions of the veins may occasionally be distinctly followed out both in the cutis and bones when these tumors are developing. 3. *Rindfleisch* claims that vascular ectasia, especially in the cavernous tumors which form in the orbital fat, is always preceded by infiltration of the tissues with small cells, which is followed by a sort of cicatricial shrinking of the tissue, and consequent tearing apart of the vessels, whose calibre must constantly be increased by continued atrophy of the intermediate tissue.

For some reasons I have long supposed that both in plexiform and cavernous angiomata there was some process similar to inflammation, but neither the latter (scarcely applicable to the cavernous tumors in bones) nor the former two hypotheses appear to fully explain the causes and peculiar differences in the distention of the vessels. We have still to mention one difference between cavernous tumors: they are either connected with the large venous trunks, as sacs to the subcutaneous veins, or numerous small arteries and veins sink into the capsule of the cavernous tissue. Lastly we must mention that these cavernous venous ectasiæ may occur accidentally in other tumors as in fibroma and lipoma, as has already been mentioned. A few years since I extirpated a lobular lipoma, which had formed below the scapula of a vigorous young man, all of the lobes of which had centrally degenerated to cavernous tissue. Cavernous angiomata develop with especial frequency in the subcutaneous cellular tissue, more rarely in the cutis and muscles, very rarely in bones, but quite often in the liver, particularly on its surface, occasionally also in the spleen and kidneys. They are sometimes quite painful, other cases are not at all so.

The *diagnosis* of cavernous angiomata is not always easy; when they occur in the cutis, they may be mistaken for more deeply-seated telangiectases, although the blood may be pressed out of the cavernous venous tumors more readily than from telangiectases. Deeply-seated tumors of this sort are always difficult to recognize with certainty; they usually show decided fluctuation, are somewhat compressible, swell on forced expiration; but the last two symptoms are not always distinct, hence they may readily be mistaken for lipomata, cysts, and other soft tumors; sometimes, indeed, this mistake cannot be avoided.

Probably half the angiomata are congenital, or at least developed soon after birth. If they develop during life, it is usually in childhood or youth; it is rare for vascular tumors to occur during manhood or old age, which is very remarkable, as the disposition to vascular diseases, especially to ectasia of the vessels, greatly increases with advanced age. Not only the larger arteries and veins dilate at this time, but also the small anastomosing vessels and capillaries, at certain localities, show visible dilatations through the skin. On the face of a ruddy, healthy old man we see red cheeks as we do in the young; it is not, however, the regular rosy bloom of a maiden's cheek, but a more bluish red, and, if you look more closely, you find numerous tortuous vessels, visible to the naked eye; in some, this redness occurs in spots. These small vascular ectasiæ do not occur in all old persons, so that we must suppose them due to a peculiar predisposition. Hence, as we said, in spite of the fact that advanced age is more disposed to disease of the vessels than any other time of life, true vascular tumors develop almost exclusively in youth. There is no doubt that the telangiectasiæ, which popularly are often called "mother's-marks," are often inherited. This appears to be proved by a number of stories about children, that have been lost, being subsequently recognized by marks inherited from the father or mother. We should undoubtedly learn far more of the hereditary transmission of vascular tumors if we would attend more to that of diseases of the vessels generally. Even if plexiform and cavernous angiomata are to be regarded as anatomically distinct from each other, and from the different varieties of varices and aneurisms, it is still clear that a predisposition to dilatation of the vessels is at the root of all of them; this is undoubtedly to a great extent inherited, and the above diseases can only be regarded as different modes of appearance of this predisposition at different ages. Hitherto attention has been so exclusively paid to the anatomical conditions of the tumors that the classes of diseases accompanying them have been too little noted.

As regards the further fate of angioma, telangiectasiæ, which are almost always congenital, may be either solitary or multiple. Their growth is always slow, painless, and is sometimes chiefly superficial again in the depth, and usually at the expense of the diseased tissue.

There is no doubt that occasionally in the course of years these tumors cease to grow, but remain unchanged. But in other cases the growth continues so that the tumors, as I once saw on the neck of a boy five years old, may grow almost as large as a man's fist. Frequently two or three telangiectases occur congenitally, or occur in quick succession, especially on the scalp, more rarely there are six or eight. I have seen two cases of flat congenital plexiform angiomata of the left side of the face, which healed at some points, partly from ulceration, partly from unknown causes; i. e., cicatricial white spots occurred here and there, where the vessels were obliterated, while in the periphery the proliferation progressed.

Cavernous angiomata are rarely congenital, but generally occur in childhood or youth, more rarely later in life. As already remarked, their seat is chiefly in the subcutaneous cellular tissue, more frequently in the face, more rarely on the trunk and extremities. They often occur in large numbers, but in such a way that a certain vascular district is to be regarded as the seat of disease, as an arm, a foot, leg, or face, etc. Besides the disfigurement, the symptoms induced are a certain weakness of the muscles, and occasionally pain in the part affected. The tumors may attain considerable size, and thus especially on the head prove dangerous, the more so, as by further progress they enter and destroy the bone. Some observations that I know of show that in these tumors, as a result of thrombosis of the cavernous spaces, there may be atrophy and retrogression (especially in the cavernous tumors of the liver); but complete disappearance of the angioma by spontaneous obliteration has not been observed.—*Treatment* for vascular tumors is very varied. The operations have two different objects:

1. Methods aiming at coagulation of the blood, with consequent obliteration and atrophy of the tumor. Among these are injecting the tumor with liquor ferri sesquichlorati; also transfixing them with hot needles, or the galvano-cautery, and drawing a platinum wire through, and subsequently heating it with the galvano-caustic apparatus (galvano-caustic setaceum). We must also mention continued compression of the tumor and ligation of the afferent artery. Both of the latter have gone out of use, as they have proved entirely worthless.

2. Methods aiming at the removal of the angioma:

(*a.*) By ligation; in telangiectasis with a broad base this must be double or multiple. A needle with a double ligature is passed through under the tumor; one ligature is tied to one side, the other to the other side of the base of the tumor.

(*b.*) In vaccinating on the tumor, so that, when the vaccine scab falls, the tumor may be removed.

(*c.*) Cauterization; for this purpose fuming nitric acid is best; it should be applied by a rod about as thick as a goose-quill, till the angioma assumes a yellowish-green color.

(*d.*) By extirpation with the scissors or knife.

After some experience in operating, the choice of these methods in any given case is not difficult. In superficial angiomata, if not altogether too extensive, and not so situated that the subsequent cicatricial contraction would cause decided deformity, as on some parts of the face, I regard cauterization with fuming nitric acid as the proper method. In extensive plexiform, and in the cavernous angiomata, removal with the knife and scissors is the most certain operation. Too profuse hæmorrhages in such operations may be prevented partly by compression of the parts around by skilled assistants, and the rapid application of the suture, partly by free mediate ligation of the whole periphery of the tumor. In many cases of angioma of the face also extirpation is to be preferred to cauterization, because the incision may be so directed that the subsequent cicatricial contraction shall induce no distortion of the eyelids or angle of the mouth. But there are cases where extirpation is entirely impracticable, partly from the size, partly from the seat or number of such tumors. I treated a child, with a still growing cavernous tumor which extended from the glabella, through the nose and whole upper lip. If it had been desired to extirpate this, it would have been necessary to remove the whole nose and upper lip; of course, this was not to be thought of; hence I tried cauterization with heated needles. The treatment had lasted three months, and would have taken as much longer, although a large part of the cavernous space was already obliterated, when the mother of the child unfortunately lost patience, and I never saw it again. I prefer this mode of cauterization to the injection of liquor ferri, as suppuration and gangrene occasionally follow the latter, and as the injection is occasionally rendered difficult by the fine canula being stopped by coagula. The other methods are of very secondary importance; vaccination frequently does not go deep enough, and the ligature is a tedious, uncertain method, which is sometimes rendered dangerous by secondary *hæmorrhage.*

[In regard to the formation of blood outside of the circulation, as an explanation of *cavernous angioma*, of late numerous observations seem to show the possibility of its occurrence physiologically (see, in regard to the placenta, Lecture XLII.) and in inflammatory and tumor new formations. In these changes there are always large cells, protoplasm masses of various natures, in which at first are formed small cells, recognizable by their coloring with hæmoglobine, which gradually acquire the size and color of red blood-corpuscles, and finally, surrounded by a fibrinous liquid, lie in a cavity composed of cells. It is probable that these cavities later become true vessels, or that at least a communication with previously formed vessels develops.

My investigations of cavernous lymphangiomata support the idea that in the immediate vicinity of the vessels, possibly in their walls, cells collect, soften in the middle, and then communicate with the interior of the vessel; we shall hereafter find a similar process in villous sarcoma. That the framework of cavernous tumors, at least sometimes, develops independently of the vascular system, is shown by the occurrence of small tumors, of cavernous formation, but not yet filled with blood, in the liver along with numerous similar tumors fully developed and communicating with the vessels.

Another method of treatment of angioma, in addition to those mentioned on page 711, is one spoken of for lupus, by making numerous fine parallel incisions through the skin by which many vessels are divided and subsequently atrophy.

Instead of using silk to ligate angiomatous tumors we may here also apply elastic ligatures. If the mark is flat and wide-spread, the incisions may first be tried, and after part of the vessels have atrophied, fuming nitric acid may be applied to the remainder of the tumor.

About 1870 the translator treated one of these tumors, affecting the lobe of the left ear and extending to the cheek, by the galvano-cautery. Two applications made at intervals of a fortnight caused its entire disappearance, only a slight scar being left. As a warning to others using the cautery about the face while the patient is etherized, it may be added that in spite of the ether having been carefully wiped away before the cautery was heated, on approaching it to the face the fumes of the ether expired by the patient at once took fire; after extinguishing the flame a paper was held between the mouth and ear to keep away the fumes while the operation was being completed.]

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In the form of an appendix I may also mention:

1. *Cavernous lymphatic tumors* (lymphangioma cavernosum), a very rare form of neoplasm, which is of the same anatomical formation as cavernous blood-tumors, but with the difference that, instead of blood, there is lymph in the mesh-work. This variety of the tumor occurs congenitally in the tongue as a form of macroglos-

sia (there is also a fibrous form); in young persons it sometimes occurs at different parts of the subcutaneous cellular tissue (lips, cheeks, chin, thigh).

2. *Nævus vasculosus*, the so-called fire-mole; this is a plexiform angioma of the most superficial cutaneous vessels, which ceases to grow from the moment of birth. There is no other difference between fire-mole and growing angioma. I have already said that there are various combinations of hypertrophy of the skin, pigmentation, ectasia of the vessels, and formation of hair in these congenital marks. If these marks be on the face, and not too large (sometimes they implicate half the face), we may extirpate them partly or entirely, and subsequently make a plastic operation, or we may resort to cauterization. Some of these marks, where only the tops of the papillæ are affected, may be greatly improved or even cured by a very superficial peeling of the skin.

## LECTURE XLVIII.

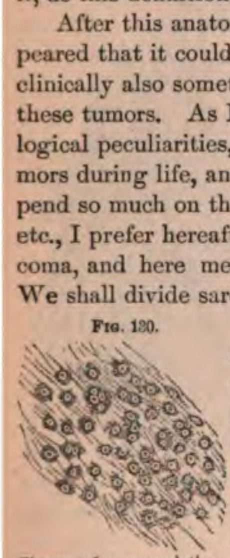
8. *Sarcomata.*—Anatomy: *a*, Granulation Sarcoma; *b*, Spindle-celled Sarcoma; *c*, Giant-celled Sarcoma; *d*, Stellate Sarcoma; *e*, Alveolar Sarcoma; *f*, Pigmented Sarcoma.—Clinical Appearance.—Diagnosis.—Course.—Prognosis.—Mode of Infection.—Topography.—Central Osteosarcoma.—Periosteal Sarcoma.—Sarcoma of the Mamma, of the Salivary Glands.—9. *Lymphomata.*—Anatomy.—Relations to Leucæmia.—Treatment.

### 8. SARCOMATA.

OVER no group of tumors has there so long been uncertainty about their anatomical position and extent as about sarcoma. The old name, taken from σαρξ, flesh, merely meant that on section the tumor had a fleshy look; of course, this did not make a diagnosis, as it was greatly a matter of choice what should be called flesh. The attempt to employ the name "sarcoma" solely for tumors composed of muscle filaments (*Schuh*), that is, to identify it with those tumors now called "myoma," was not popular. Subsequently the term became somewhat more definite, as it was made to include all tumors rich in cells which had no decided alveolar formation, and were not carcinomatous. It is only for the last ten years that the following histological definition has received general acceptance and has become quite common. A sarcoma is a tumor consisting of tissue belonging to the developmental series of connective-tissue substances (connective tissue, cartilage, bone), muscles, and nerves, which, as a rule, does not go on to the formation of a perfect tissue, but to peculiar degenerations of the developmental forms. Some pathologists would gladly see "muscles and nerves" excluded from this definition, but when speaking of spindle-celled sarcoma I shall show why I cannot admit this. If it is desired to term the inflammatory neoplasiæ in their various stages examples of sarcoma (*Rindfleisch*), I assent to it, as this definition would agree pretty well with mine.

After this anatomical basis was found for "sarcoma," it soon appeared that it could be diagnosed, even with the naked eye, and that clinically also something could be said about the peculiar course of these tumors. As I think that the subdivisions, according to histological peculiarities, are less important for the diagnosis of these tumors during life, and that their diagnosis, prognosis, and course, depend so much on their point of origin, the rapidity of their growth, etc., I prefer hereafter classing together the clinical remarks on sarcoma, and here merely considering more attentively the histology. We shall divide sarcoma into the following forms:

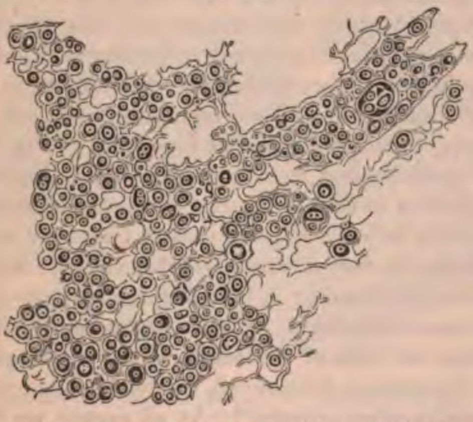
FIG. 130.



Tissue of a granulation sarcoma. Magnified 350 diameters.

(*a.*) *Granulation sarcoma, round-celled sarcoma* of *Virchow.* This tissue is the same, or very like that of the upper layer of granulations; it always contains chiefly small round cells, like lymph-cells; the intercellular substance is sometimes scarcely perceptible, again it is in greater quantities, and may be perfectly homogeneous, as in neuroglia (*Virchow's* glioma and glio-sarcoma), or it is slightly striated (Fig. 130), or even fibrous, or may be œdematous (as in large mammary sarcomata). Lastly, it may also be reticulate, and so approximate the tissue of lipoma.

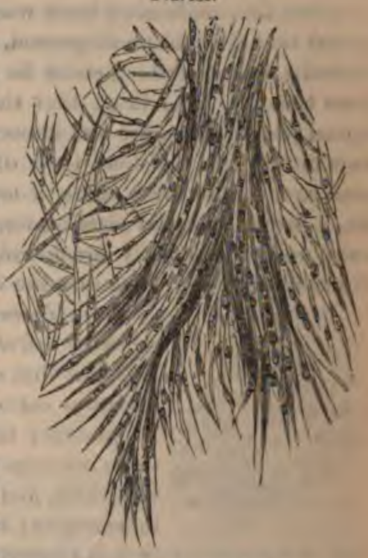
FIG. 131.



Tissue of a glio-sarcoma, after *Virchow*. Magnified 350 diameters.

(*b.*) *Spindle-celled sarcoma* is composed of closely-packed, usually thin, elongated spindle-cells, so-called filament-cells. Usually there is no intercellular substance, occasionally there is some, it may be homogeneous and soft, or fibrous; if the fibrous portion preponderates, the tumor is called fibro-sarcoma, or fibroma. Formerly this spindle-celled tissue was termed young connective tissue (tissue fibroplastique, *Lebert*); but from my histogenetic investigations in the embryo I have long protested against this view, for spindle-celled tissue, as we usually find it in these sarcomata, does not occur in embryonal tissue at any period, not even in the tendons; the physiological example of this tissue is young muscle and nerve tissue; these spindle-celled sarcomata would then be young myomata or neuromata. *Virchow* has carried the same view further, especially as far as regards fibrous uterine tumors (page 685). I protested against this view of *Virchow's*, with its consequences, as the diagnosis is always doubtful in special cases. When a nerve contains a tumor consisting of elongated spindle-cells, whose ends terminate in fine filaments, it is very natural to regard it as a neuroma whose elements are not fully developed at any point. When a spindle-celled tumor is developed in muscle, and the fibre-cells show band-like forms, even fine granulation, as in the commencement of striation, there could be no blame for calling these tumors "myomata," under the idea that they were young muscle-tissue that had not gone beyond certain bounds of development. So far there is no objection to this view. But when a spindle-celled sarcoma comes in the cutis, or on the penis (where I recently saw a remarkable case), we may be very doubtful whether the case is one of young neuroma, myoma, or fibroma; in both of these parts there are nerves, muscles, and connective tissue. If, then, there be nothing typical in the arrangement or form of the cells, and the histological mode of origin cannot be certainly determined, we must content ourselves with the term "spindle-celled sarcoma." At all events, we have to deal with a fibrous tissue, whose development has not advanced beyond the production of spindle-cells. Moreover, I think I can affirm from my observations that the course and prognosis of these tumors scarcely

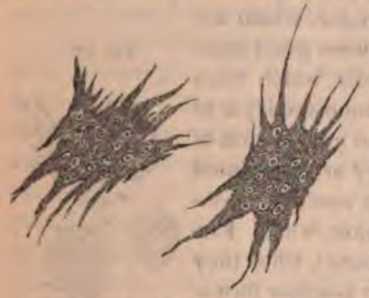
FIG. 132.



Tissue of a spindle-celled sarcoma.

depend on their origin, but far more on their locality, rapidity of growth, consistence, and other clinical conditions.

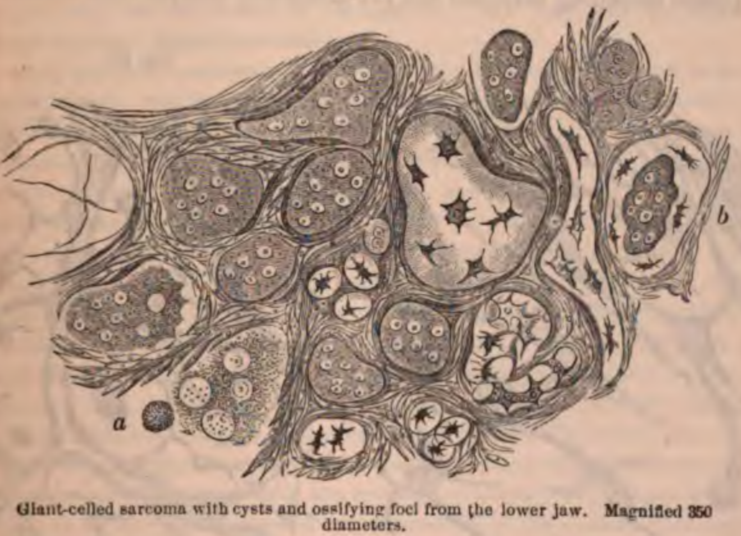
Fig. 133.



Giant-cells from a sarcoma of the lower jaw. Magnified 350 diameters.

(c.) *Giant-celled sarcoma* is a name given by *Virchow* to a variety of sarcoma containing very large cells, which are partly round, partly polymorphous, and supplied with many offshoots (Fig. 133). These cells, which normally occur in the medulla of the bones of the fœtus, although not so large as in tumors, have excited great astonishment by their size; they are the largest unformed protoplasm collections that have been seen in man; they may contain thirty or more nuclei, and their origin from a simple cell by a series of transformations is generally easily followed. These giant-cells occur in spindle-celled, as well as in fibro-sarcoma; they occur somewhat smaller sporadically, and are also found in granulation and myxosarcomata. They are most frequent in the central, less so in periosteal sarcoma, but I have seen them even in muscle-sarcoma. By their size they occasionally give

Fig. 134.

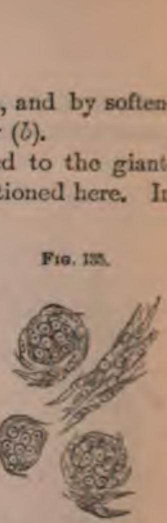


Giant-celled sarcoma with cysts and ossifying foci from the lower jaw. Magnified 350 diameters.

the tissue an apparently alveolar (Fig. 134) structure, and by softening may lead to formation of cysts (*a*), or may ossify (*b*).

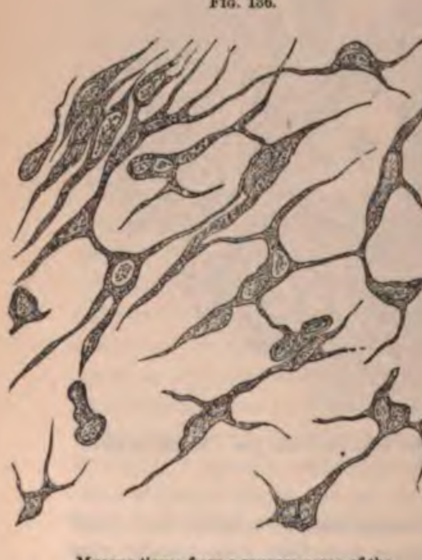
A peculiar formation from sarcoma, which is allied to the giant-cell, although never growing very large, may be mentioned here. In a granulation-sarcoma of the dura mater, which accidentally fell into my hands, there were great numbers of globular, multinucleated cells, which were surrounded with a membrane-like connected layer of spindle-cells (Fig. 135). I hazard no explanation of these elements, but suspect that they are associated with the formation of tufts on the cerebral membranes, and with tufted fibro-sarcomata, which *Virchow* calls brain-sand tumors (psammone), when they contain brain-sand. Possibly, these peculiar formations are aborted excrescences from blood-vessels, an idea I have long cherished, and which is apparently confirmed by a recently-published observation of *Arndt*, who saw these spheres attached to vessels by pedicles. *Waldeyer* lately showed that these and allied formations, which occur especially in intracranial tumors) start from the perithelial (adventitial) cells of cerebral vessels. The neoplasiæ belonging here, but not yet sufficiently analyzed and classified,

FIG. 135.



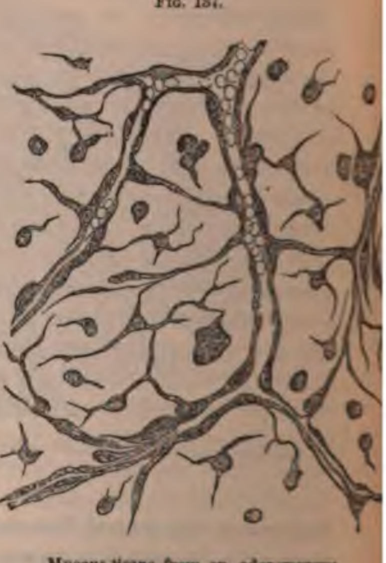
Cell-globules from sarcoma of the du mater. Magnif 350 diameters.

FIG. 136.



Mucous tissue from a myxosarcoma of the scalp. Magnified 400.

FIG. 137.



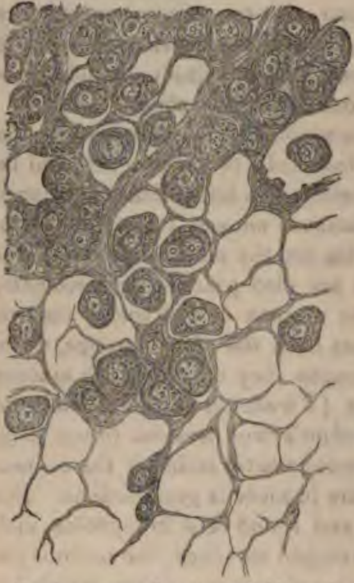
Mucous tissue from an adenomyxoma of the mamma. Magnified 400.

as well as the alveolar sarcomata of which we shall soon treat, often so much resemble carcinomata in their structure that they are very difficult to distinguish. According to recent observations, especially those of *Sattler*, what I formerly described as cylindroma, and erroneously classed with adenoma, also belongs in this class.

(*d.*) *Net-celled sarcoma. Mucous sarcoma.* (Gelatinous sarcoma of *Rokitansky.*) For the offshoots from cells to develop well and be distinctly seen, there must be considerable soft intercellular substance present. Hence sarcomata with gelatinous mucous intercellular substance, which contain any stellate cells, are the most beautiful. But this is not always the case. There are also granulation-sarcomata, that have a claim to be regarded as mucous or gelatinous tumors. If we should wish to class the tumors from the above groups, when they appear gelatinous, together because they contain much mucous (μυξα), we may call them myxomata (*Virchow*), or retain their old name, collonema (*J. Müller*). *Virchow's* true mucous tissue (Fig. 135) undoubtedly belongs to the developmental series of the connective tissues; occasionally it also occurs in mucous granulations. But frequently also we find spindle-cells and round cells in myxoma, and, if there be at the same time any developed cartilage, the mucous tissue may be regarded as young or softened cartilage-tissue, which becomes the more probable if a myoxoma contains honey-comb-like septa such as are found in chondroma. We may use the terms myxosarcoma, myxochondroma, etc.

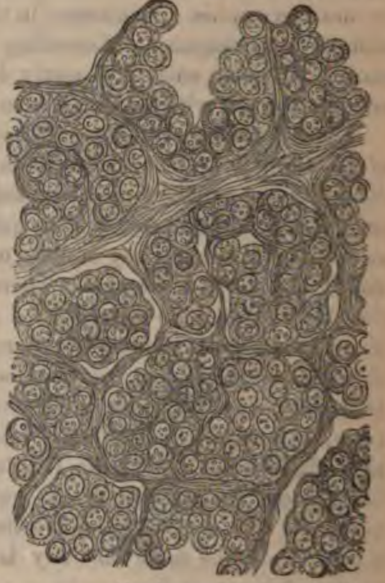
(*e.*) *Alveolar sarcoma.* This rare form of tumor (occurring in the cutis, muscle, and bone) is very difficult to characterize anatomically; from the size and arrangement of its cells, it may in spots so much resemble carcinoma, that I would not trust myself to decide correctly on every piece of such a tumor placed under the microscope. The cells of these elements are much larger than lymph-cells, about the size of cartilage-cells, or of moderately large flat epithelium, and usually have one or more large nuclei, with glistening nucleoli. The cells are embedded in a fibrous, or more rarely homogeneous, slightly-developed intercellular substance of exquisite alveolar type, in such a way that they lie together separately, or more rarely in groups (Figs. 138 and 139). They are most intimately connected with the fibres, and are difficult to detach from the fibrous mass. The latter two peculiarities are important for the histological diagnosis of "sarcoma," for they show the large cells are connective-tissue cells, not epithelial cells, as in true carcinoma-tissue. Occasionally the cellular elements of these sarcomata lie in immediate contact, without any intercellular substance; the resemblance to epithelial carcinoma may prove deceptive. *Virchow* has described and deduced this form from soft warts of the cutis.

Fig. 138.



Alveolar sarcoma from the deltoid muscle. Magnified 400 diameters.

Fig. 139.



Alveolar sarcoma from the tibia. Magnified 400 diameters.

(*f.*) *Pigmentary sarcoma. Melanotic sarcoma. Melanoma.* All these names indicate pigment formation in sarcoma. This pigment, which is usually granular, rarely diffuse, is brown or black, lies almost always in the cells, rarely in the intercellular substance. Part or the whole of the tumor may be faintly or distinctly black. Any of the above forms of sarcoma may occasionally be pigmented, but I have most frequently found this to be the case in the last form, and in the spindle-celled sarcoma. Melanomata develop most frequently in the cutis, especially of the foot and hand, but also on the head, neck, and trunk.

The arrangement of the cellular elements in sarcoma depends, on the one hand, on certain directions of the fibres or fibre-cells in the tissue of the tumor; on the other, on the form of the vascular network; from these circumstances, as well as from the development of giant-cells, or similar formations, there may result an arrangement of the tissue of the tumor, scarcely distinguishable from the areolar formation formerly ascribed exclusively to carcinoma-tissue. This should not astonish you, for in cartilage also we have a type of cavities with enclosed cells, and also the net-work of the lymphatic glands, which undoubtedly belong to the system of connective-tissue substances, but must also be termed alveolar formations.

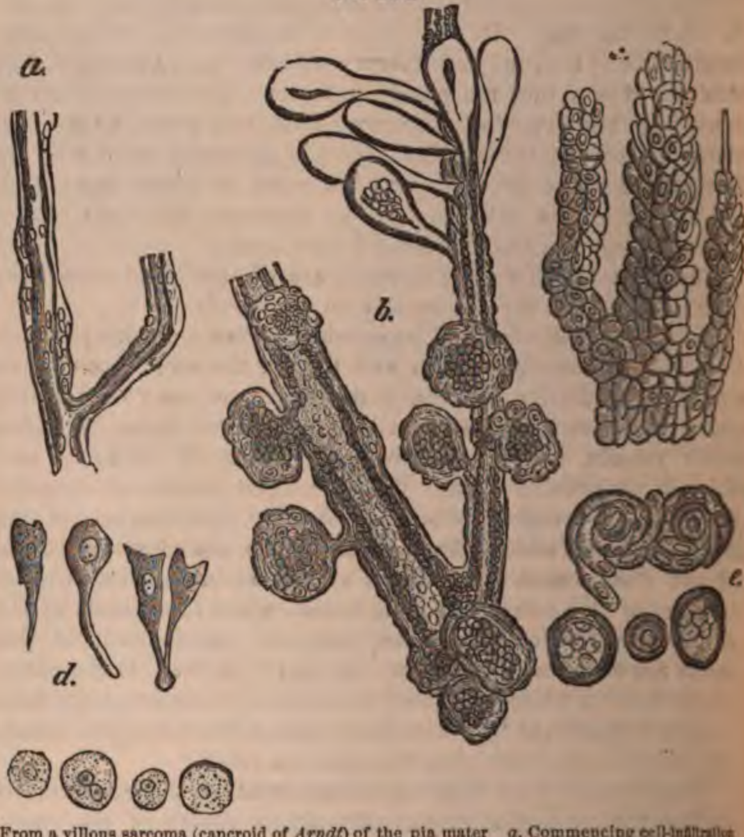
[To return a moment to (*d*): the large cells found normally in the medulla of fetal bones were termed by *Robin* "myèloplaxes," and giant-cells, from their resemblance to these, have occasionally received the same name. Giant-cells are often closely related to the formation of vessels, in whose vicinity they usually lie, as round cells, derived from normal connective tissue, whose protoplasm increases, probably at the expense of neighboring cells, and the nuclei multiply. In this phase of development they are true hæmatoblasts; i. e., the protoplasm partly changes to hæmoglobine, which divides up into red blood-corpuscles. The intercellular substance near the giant-cells becoming fluid, may form cavities containing blood-corpuscles (blood-cysts). Although giant-cells are not confined to the above tumors, but occur in spindle and round-celled sarcoma, yet here they are so numerous and large that a variety of sarcoma has been named from them.]

The following forms of sarcoma are of developed connective tissue, whose form depends greatly on the vessels:

(*g*.) The (infiltrated and superficial) *villous sarcoma*, *pearl-tumors*, and *psammona*. As is well known, the serous membranes have the peculiarity in some pathological processes of forming ragged proliferations, whose basis is connective tissue and ultimately vessels, and whose covering consists of multiplied and enlarged endothelial cells. Well-developed shreds of synovial membrane in arthritis deformans, shreddy proliferations of the pericardium and endocardium on the valves, the plexus choroidei, and the Pacchionian granulations of the cerebral membranes, are the types of this neoplasia. The tumors which to a certain extent may be regarded as the highest stages of development of this variety are only found in the membranes of the brain or the nerve-sheaths directly proceeding from it; some of these neoplasiæ have a villous character, at least exteriorly; others form compact masses, the dendritic tissues growing through each other.

These tumors form thus: A circumscribed cellular infiltration (*a*, Fig. 139 A) begins in the adventitious sheath of the vessel, which gives rise to clubbed, shreddy outgrowths, which soon become hyaline or filamentary connective tissue, and then develop a cavity in them, which gradually unites with the caliber of the vessel (*b*). Part of the cells assume epithelial forms and envelop the above club-shaped neoplasiæ (*c*). Between these cell-masses we find spheres of flat compressed cells (*e*), which in part become dry and under some conditions even calcareous. Whether the *pearl-tumors* (*Virchow*) starting from the cerebral membranes, which are composed of pearly non-vascular nodules from the size of a millet-seed to a pea, are composed of such endothelial spheres, or are true epithelial formations, I shall not attempt to decide, as I have made no personal observations, and there has been nothing published on this point recently. According to *Virchow's* investigations, the pearls of the intracranial tumors are composed of connective-tissue cells; hence they should be classed with sarcomata. Thymus pearls are the physiological examples of this form, which from their non-vascularity also have an analogy to tubercle.

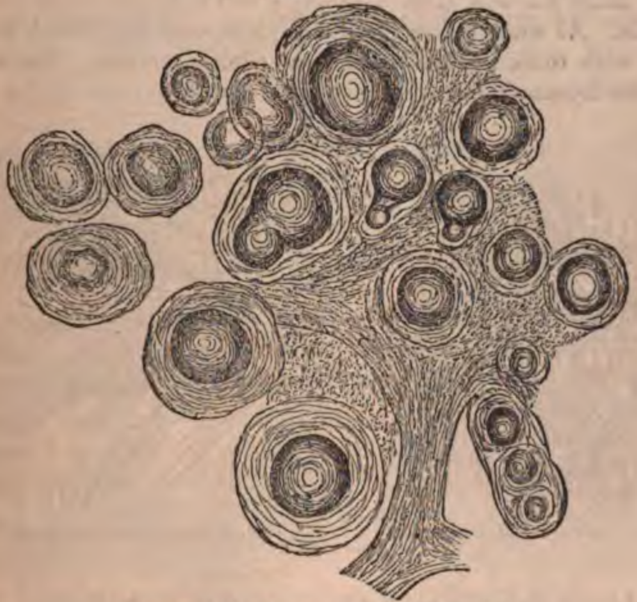
FIG. 139 A.



From a villous sarcoma (cancroid of *Arndt*) of the pia mater *a*, Commencing cell-infiltration in the capillary walls; *b*, clubbed proliferations growing from the walls of the vessels; *c*, the same covered with a thick layer of endothelium; *d*, endothelial cells of the highest development, not to be distinguished from epithelial cells; *e*, conglomeration of these cells into a spherical shape. Endothelial pearls. Magnified 400.

Another tumor described by *Virchow* and renamed belongs here, the *psammona*. This also has only been observed in the brain or in the orbit, and is related to the villous and to plexiform sarcoma, which we shall soon describe. This variety of tumor is characterized by the occurrence of calcareous spheres, having the form of the concrements which are found in the pineal gland, and are there known as brain-sand (πσαμμός, sand). Like the thymus pearls, these are mostly connected with the vessels, and are probably mostly calcified endothelial pearls ; but *Virchow* says that direct calcification of connective tissue may lead to the same forms.

FIG. 139 B.

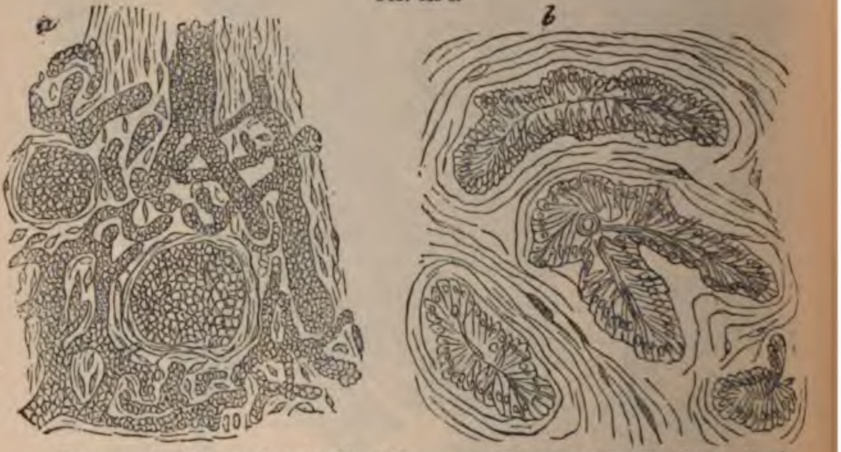


Psammona, after *Virchow*. Magnified about 200.

(*h.*) Plexiform (cancroid, adenoid) sarcoma. This form of sarcoma also is chiefly found in the orbit and brain, but sometimes occurs in the parotid gland. It can only be distinguished by very careful examination from some forms of carcinoma to be hereafter described. Plexiform cylinders, clubs, and spheres of small cells spread out in the connective tissue, separating its bundles and filling all the interspaces between them, in doing which they naturally push into the lymphatic vessels and perivascular lymphatic spaces. It cannot always be determined whether the cells first increasing are wandering cells, connective-tissue cells, or cells from the walls of the vessels, endothelium, or perithelium ; perhaps they all participate at the same time or after one another.

[Development of the tumor starts from the blood-vessels, which, by proliferation of their endothelium, become tubular, netlike, anastomosing structures, full of small, round, or irregular cells, embedded in a close, fibrillar connective tissue. The cells enlarge, their borders become indistinct, finally they so fill the tube that its caliber is no longer perceptible; the nuclei of the cells originating from the endothelium remain, and distinguish the tubuli from true epithelial gland-tubes. While the endothelium has increased in size, the rest of the vascular wall has become hyaline, so that in the tubes there is a wall (tunica propria) and a distinct inner coating of cells. At some places occur irregular, nodular, villous formations, with thick hyaline walls and cellular contents. Elsewhere from the hyaline sheath papillary offshoots project among the cells, and, in sections, these appear as hyaline spheres, surrounded on all sides by cells. The enveloping cells have such an exact epithelial form as to be mistaken for gland-tissue, especially if not highly magnified. From the prominent participation of the vessels in the formation of such sarcomata, this group of tumors has been called "angiosarcoma" (*b*, Fig. 139 c). These are not to be confounded with cavernous, telangiectatic sarcoma, which are characterized by very great vascularity.

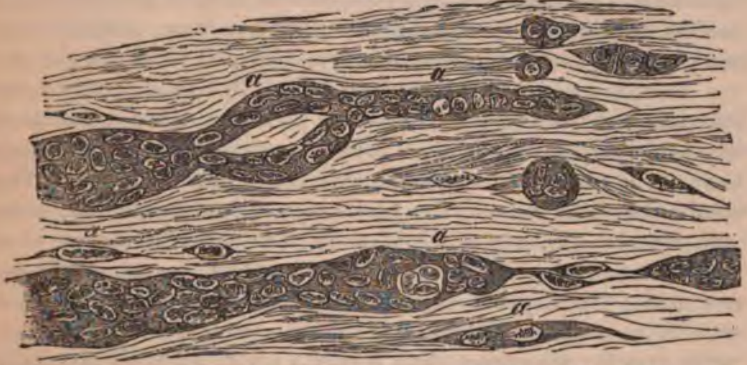
FIG. 139 c.



*a*, From a cerebral tumor, after *Arnold*. *b*, From a cerebral tumor, after *Rindfleisch*. Magnified 300–400.

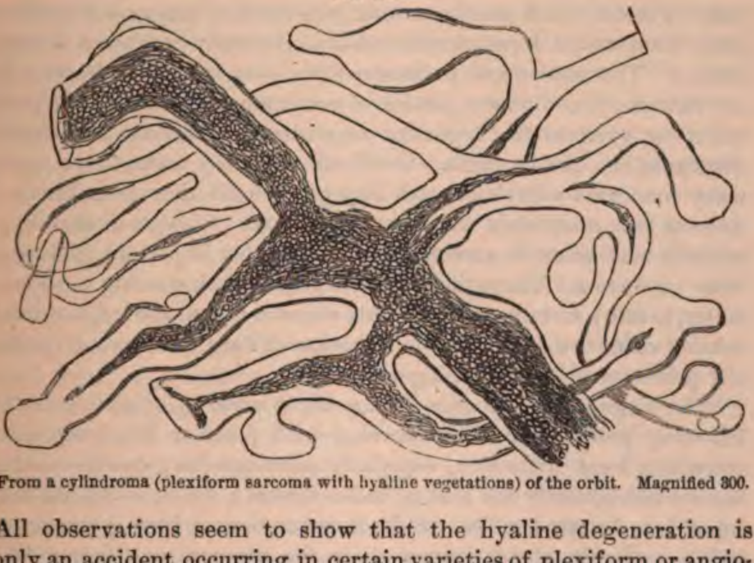
The hyaline metamorphosis sometimes affects also the cells in the tubes; it starts from the nuclei, and, by transformation of the protoplasm, branched, cactus-like, hyaline formations are thus developed. As the degeneration begins in the center, the hyaline cylinders are often found enveloped in cells. The same process is witnessed in cell-groups formed by proliferation of connective-tissue elements. These peculiar hyaline nodules and cylinders (Figs. 139 D, 139 E) were at first regarded as lymphatic vessels; then it was thought the cell-tubes contained gland-elements. From the hyaline cylinders the tumors were called "cylindroma." *Sattler's* investigations first fully explained the development of these tumors.

FIG. 139 D.



Commencing hyaline metamorphoses in the early stages of a plexiform sarcoma. Commencement of the formation of cylindroma. After *Sattler*. Magnified 500.

FIG. 139 E.



From a cylindroma (plexiform sarcoma with hyaline vegetations) of the orbit. Magnified 300.

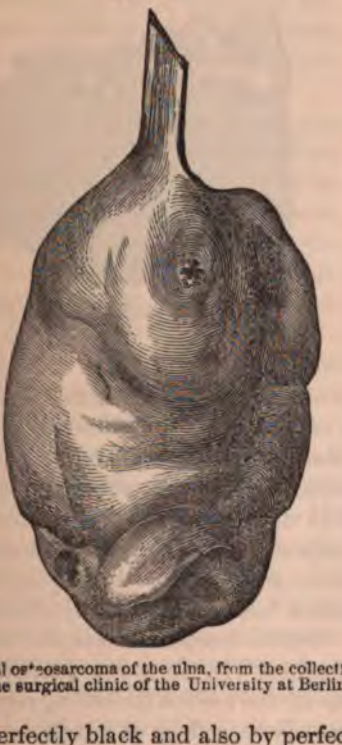
All observations seem to show that the hyaline degeneration is only an accident occurring in certain varieties of plexiform or angiosarcomata.]

Coming now to the symptoms of sarcoma perceptible to the naked eye, we must first state that in most cases these neoplasiæ have a roundish, sharply-bounded form, indeed, are usually distinctly encapsulated; this is a very important distinguishing mark from infiltrated carcinoma. Sarcoma very rarely appears on surfaces (whether free or sac-like membranes) in a papillary or polypous form; still, there are non-glandular nasal and uterine polypi, also soft warts on the skin and mucous membrane, which, from their histological structure, can only be classed among the sarcomata. The consistence and color of sarcomata vary so much that nothing general can be said about them; they may be as hard as cartilage, or of gelatinous, nearly fluid consistence. On incision, the tumor may appear bright red, white, yellowish, brown, gray, black, dark red, and different shades of all these colors may appear on the same cut surface, apart from the pigmentation; this depends especially on their vascularity, and on more or less recent extravasations of blood in the tumor. The vascularity varies greatly; sometimes there is only a scanty net-work of vessels; again, the tumor is like a sponge, traversed by cavernous veins. We must here mention another peculiarity of sarcoma: it is occasionally so white that, if it be soft at the same time, it greatly resembles brain-matter. This *medullary sarcoma* (encephaloid) usually has all the malignant qualities of sarcoma in the highest grade, and is much feared; it may have any of the above-described histological characters. Tumors which may be torn up into bundles in certain directions have been called sarcoma fasciculatum (formerly carcinoma fasciculatum). The anatomical metamorphoses that take place in sarcoma are various: the different modes of softening predominate; mucous softening, even to the formation of mucous cysts, fatty and cheesy degenerations, are frequent. Ossification is very common in sarcomata connected with bone, and may go on until the whole tumor is more or less completely transformed to bone. Cicatricial shrinkage scarcely ever occurs in sarcoma; this is another important difference from carcinoma. Ulceration from within outward, opening out like a crater, is rare; sarcomata of the cutis ulcerate early, without, however, causing extensive destruction; ulceration of hard sarcomata occasionally produces well-developed granulations.

The diagnosis of sarcoma during life is made by attending to the following points: Sarcomata develop with peculiar frequency after precedent local irritations, especially after injuries; cicatrices, also, are not unfrequently the seat of these tumors; black sarcomata may come from irritated moles. Skin, muscles, nerves, bone, periosteum, and, more rarely, glands (among these the mamma most frequently), are the seats of these tumors. Sarcomata are rarest in children, rare between ten and twenty years, most frequent in middle life, and rarer again in old age. According to my observation, men and women are affected with equal frequency. If these tumors be not located in or on nerve-trunks, they are usually painless till they break out. If the sarcoma be in the subcutaneous cellular tissue or in the breast, it may be felt as an encapsulated movable tumor. The growth is sometimes rapid, sometimes slow; the consistence varies, so that it can scarcely be used as a point in diagnosis.

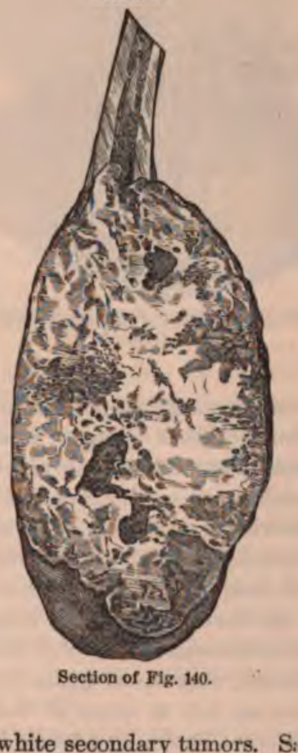
*Course and prognosis.* A sarcoma may develop solitarily, may remain so, and never return after operation. It may develop as solitary or multiple, and return after repeated extirpation; metastatic tumors may form in the lungs or liver, and thus this disease may cause death in three months. You see that the greatest benignity and greatest malignity may be united in this one group of neoplasia; indeed, I can assure you that two sarcomata of the most similar histological qualities (usually, however, with different consistence) may differ entirely in course. From this circumstance the greatest objections have been made to pathological histology; it must be acknowledged that the histological structure of a tumor by no means corresponds to its clinical course; but for this reason to cast a slur on anatomy would be just as strange as to blame it because we cannot certainly distinguish between the microscopic preparations of a salivary, lachrymal, or mucous gland, although they play very different parts in the organism. We must first overcome the habit of seeking specific anatomical forms for specific functions. But there is no lack of indications for prognosis in regard to any sarcoma. We shall hereafter speak of the importance in this respect of the location of the tumor; the consistence is important, firm sarcomata are of better prognosis than soft ones; alveolar forms are of especially bad prognosis, and still more so are the soft granulation and spindle-celled sarcomata, which usually appear in the medullary form; black sarcomata are also especially dangerous, the firm ones being less rapid in their course than the soft. The rapidity of the growth first appearing is very important for the prognosis; this is, moreover, in proportion to the consistence; if a sarcoma has taken four or five years to attain the size of a hen's egg, the prognosis is not so bad; if in four or five weeks it has grown to the size of a fist, it is very bad. A sarcoma may be mistaken for a cold abscess; I know of one case where a sarcoma of the abdominal walls developed so rapidly that at first it was diagnosed to be furuncle. In a few months the patient was covered with sarcomata, and, in less than three months from the development of the first tumor, she died from the disease attacking the lungs. Sometimes, however, a slowly-growing, firm sarcoma is followed by one of rapid growth, but the reverse of this never occurs. Usually, sarcomata develop in strong, well-nourished, often in particularly healthy and fat persons; I saw a medullary sarcoma of the mamma in a blooming, strong, healthy girl eighteen years old; she died of sarcoma of the lungs a few months after operation. The mode of development of sarcomata which appear successively is very characteristic. The first tumor is completely extirpated; after a time, in, under, or near the cicatrix, a new tumor appears; this also is completely removed; again, a new tumor appears at the point of operation, or at a slight distance from it, and near it other new ones; the patient begins to emaciate; possibly further operations are not practicable, marasmus occurs, possibly lung or liver tumors, with their symptoms, develop; the patient dies from suppuration from the primary tumor, or from disease of internal organs. The course just described differs from that of carcinoma, because in the latter continuous recurrence is the most frequent, while in sarcoma the regional predominates, provided the tumor has been entirely extirpated. This may readily be explained by the fact that the bounds of infiltrated carcinoma are much more difficult to determine than are those of encapsulated sarcoma: hence, *ceteris paribus*, the latter may be more certainly removed; if portions of sarcoma be left, of course there will be continuous recurrence. After complete extirpation of sarcoma, years may elapse before the regional recurrence, and sarcoma may always remain a local trouble for years, possibly till death. I know one case of fibro-sarcoma of the back of the head, where it was twenty-three years from the development of the first tumor till death from recurring tumors; meantime, the patient was operated on five times, and, on each occasion, he was cured for some time. From an old woman I extirpated a medullary sarcoma (alveolar cancerous form, Fig. 138) from the deltoid muscle; the wound had scarcely healed when a new sarcoma, like the first, formed in it; now the woman remained perfectly well four years, then a new tumor came in the deltoid; it was removed by an operation, probably imperfect, and recurred in the incomplete cicatrix; exarticulation of the arm was followed by recurrence in the pectoral and latissimus muscles, and death from sarcoma of the lungs and pleurisy. A year since, I extirpated a melanotic, large-celled sarcoma from the scalp of an old man, from whom *Schuh* had, six years previously, removed a similar tumor; up to the present time there has been no recurrence. When we amputate the thigh for sarcoma of the leg, after years it may recur in the amputation-cicatrix, and be followed by sarcoma of the lungs. The local tendency to recur could be explained by an extensive sprinkling of seed in the vicinity of a tumor, if the recurrences succeeded each other rapidly, but, when years elapse between the recurrences, this explanation will hardly answer, for it is not very probable that tumor cells would lie quiet in the tissue for years, and then suddenly shoot out like an old seed. I know no explanation for this mode of recurrence. The course of the infection is very peculiar in sarcoma; I think I was one of the first to show that it is an essential peculiarity of sarcoma, that it does not attack the lymphatic glands, or does so quite late in the disease. The course of sarcoma-infection goes chiefly, if not exclusively, through the veins—not, as in carcinoma, through the lymphatic vessels. Sarcomata of the lungs are mostly of embolic origin; it seems that the walls of the veins in sarcoma are very readily traversed by the tumor-substance, and their calibre filled with friable masses of it, which thence pass into the lungs. The number of the secondary sarcomata is often enormous, the whole pleura and peritonæum may be covered with them. In this respect, the melanotic forms almost appear to dispute the precedence with the medullary. Primary, only partially-pigmented tumors are occasionally followed by perfectly black and also by perfectly white secondary tumors. Sarcomata of the lungs are almost always of the granulation variety. In

FIG. 140.



Central osteosarcoma of the ulna, from the collection of the surgical clinic of the University at Berlin.

FIG. 141.



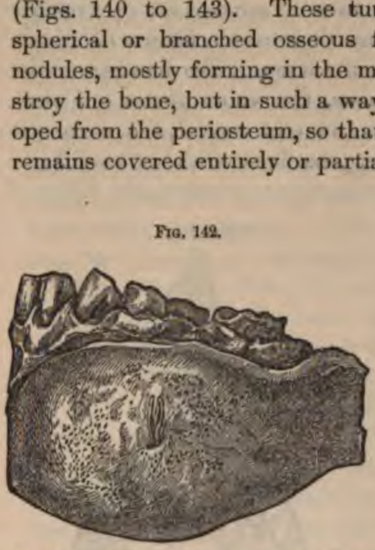
Section of Fig. 140.

the liver I have seen secondary, very beautifully pigmented, spindle-celled sarcomata; the forms of primary and secondary sarcomata thus vary greatly.

*Topography of sarcoma.* As the above general remarks are insufficient for practice, we must study more accurately different forms of sarcoma in certain tissues and in certain parts of the body.

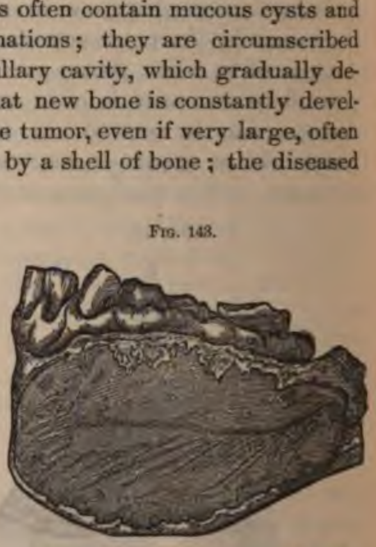
Sarcomata occur quite often in hollow bones (myeloid tumors or central osteosarcoma), usually in the form of giant-celled sarcoma; they especially attack the lower jaw, next the tibia, radius, and ulna (Figs. 140 to 143). These tumors often contain mucous cysts and spherical or branched osseous formations; they are circumscribed nodules, mostly forming in the medullary cavity, which gradually destroy the bone, but in such a way that new bone is constantly developed from the periosteum, so that the tumor, even if very large, often remains covered entirely or partially by a shell of bone; the diseased bone then appears puffed up like a bladder, and the tumor does not always cause a complete solution of its continuity. When these sarcomata occur in the lower extremity, they become very vascular; numbers of small traumatic aneurisms develop in them, and a true aneurismal murmur may be heard in them, so that they are often considered and described as true bone-aneurisms. The cystosarcomata and compound cysts, which are occasionally seen in bones, especially in the lower jaw, also in large hollow bones, have usually developed from osteosarcomata (Fig. 144). Central osteosarcomata are usually solitary, very rarely generally infectious. In the lower or upper jaw they are apt to come at the time of the second dentition, rarely at the first: in the long bones I have only seen them at middle age; of the tumors called epulis (the word means located on the gums) a

Fig. 142.



Central osteosarcoma of the lower jaw of a girl nine years old.

Fig. 143.

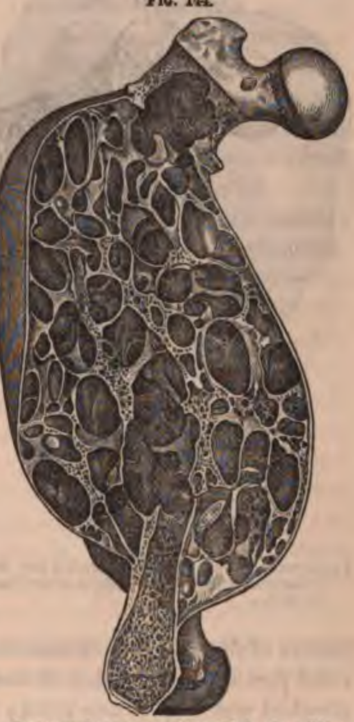


Section of the specimen shown in Fig. 141.

large number belong to these giant-celled sarcomata; their location on the gums is generally only apparent; they usually spring from cavities in the teeth, and have started from carious roots of teeth. Some also call epithelial cancer epulis; it is well either not to use such terms or to restrict them by certain adjectives; as sarcomatous, fibrous, carcinomatous epulis, etc. Peripheral osteosarcomata or periosteal sarcomata (osteoid-chondromata of *Virchow*) are quite malignant; they either have granulation structure with osteoid tissue as in osteophites, and are partly ossified; or they are very large-celled myxosarcomata, also partly ossified. The rapidity of the course varies greatly; sarcomata of the lungs have been observed after them.

Spindle-celled sarcomata are found especially often in muscles, fasciæ, and cutis; they are locally very infectious, and often return after extirpation. Myxosarcomata come in the cutis and subcutaneous cellular tissue, and with the naked eye are often difficult to distinguish from œdematous soft fibromata. The nerves also are relatively often the seat of multiple sarcoma. The more rapidly the primary tumors have grown, and the more "medullary" their appearance, the more dangerous they are. I find that all ages, except perhaps childhood, are equally disposed to these tumors.

FIG. 144.

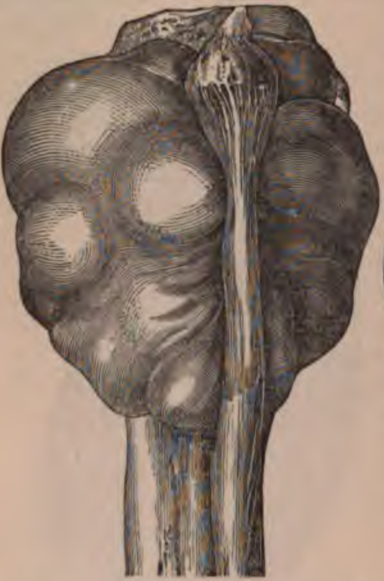


Compound cystoma of the thigh, after *Péan*

When sarcoma develops in a gland it almost always contains glandular elements, which may be greatly changed in form, and some of which may be newly formed. Hence, pure adenomata (which are very rare) may be difficult to distinguish from sarcomata that have developed in glands (adeno-sarcomata). Glands are by no means equally disposed to the development of sarcoma; we shall briefly state the localities where they are most frequently found.

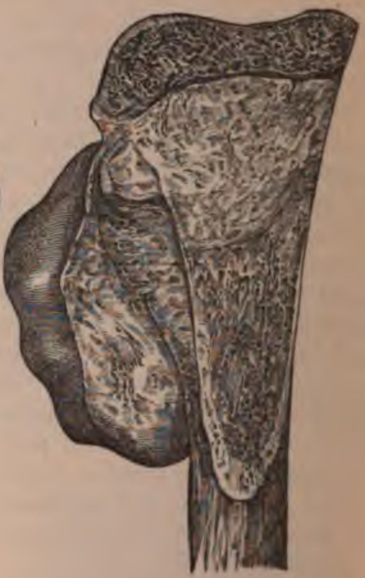
The female mamma, more than any other gland, is subject to these tumors. Sarcomata of the mamma are roundish, lobular, nodulated

Fig. 145.



Periosteal sarcoma of the tibia from a boy, from the collection at the surgical clinic of the University at Berlin.

Fig. 146.



Section of Fig. 146.

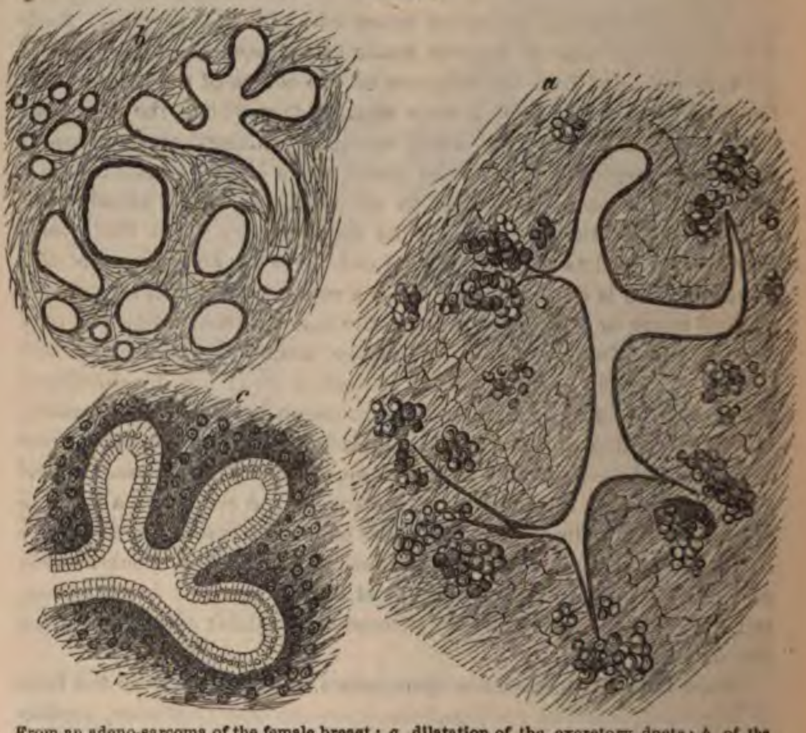
tumors of firm, elastic consistence; the disease may attack a large or small portion of the lobes of the gland; as a rule, only one breast is attacked and only at one point; at other times, several small nodules occur at the same time in one gland. These tumors grow very slowly, cause no pain; like all sarcomata, they are sharply bounded from the healthy parts, hence they are movable in the glandular parenchyma; when they grow large (in the course of years they may attain the size of a man's head) they almost always form cystosarcomata; in the course of time they become softer and cause pain; ulceration also occurs. The anatomy of these tumors has always excited great interest. As the glandular elements, acini as well as excretory ducts, were found in them, it was formerly supposed that they had developed in the tumor; hence these tumors were called partial hypertrophies of the mamma. I consider this view incorrect, and think that, by examining a great many of these tumors, I have satisfied myself that primarily and chiefly there is a development of sarcoma in the connective tissue around the acini, the latter being preserved, although they may be changed in various ways. The distention of the gland-ducts causes cysts, at first slit-shaped, subsequently more roundish, with muco serous contents, whose development we shall immediately follow. The tissue of the neoplasia itself is usually composed of small, round, spindle-shaped, rarely of branched cells, with considerable developed, fibrous, sometimes gelatinous intercellular substance. In some of these tumors the fibrous tissue may be so prevalent that, in consistence and constitution, the entire tumor may resemble fibroma. Accidental cartilaginous and osseous tissue are occasionally observed, but are very rare, and have no influence on the course of the disease. If the growth of these tumors were regular throughout, the excretory ducts and acini of the glands would be equally enlarged or compressed; for, if you imagine a part of the gland, say a lobule, spread out as a surface, and suppose the basis to which this surface is attached enlarging, the epithelial surface must also enlarge. But the glands may be regarded as surfaces bulged out in many places, so that this representation is quite proper. Such a regular growth in all parts of a gland never or very rarely occurs; the result is, that frequently only the excretory ducts elongate or enlarge much; this induces the slit-shaped, elongated cysts, visible to the naked eye; but, by simultaneous distention of the glandular acini, roundish cysts are often formed. In this stretching of the sacculated glandular surface, the epithelium increases and develops to a higher stage, inasmuch as the small, round epithelial cells of the acini increase greatly, and change to a layered-cylindrical epithelium. The glandular substance thus altered secretes a muco-serous liquid, a very minute portion of which is spontaneously evacuated from the nipple, while most of it is retained in the tumor, and serves to dilate the already distended glandular cavity (retention and secretion cysts).

Then the tumor-substance again grows into these cysts in the form of lobulated, leaf-like proliferations (cystosarcoma phyllodes, proliferum; *John Müller*), so that the cut surface may thus acquire quite a complicated appearance.

The relation of this cyst-development to the sarcoma (the nature and course of the disease is not much influenced by the former) varies greatly in these, as in all cystosarcomata.

Mammary and cysto sarcomata are not very rare, but are far less frequent than the cancers of the breast, which we shall hereafter mention. The disease is most frequent in young married women, but also occurs shortly before puberty—rarely after the fortieth year of life. The growth of these tumors is very slow, and is painless before they become large; later, however, they are accompanied by piercing pains; as the tumor may grow as large as a man's head, and ulcerate, it may prove very troublesome. Some of these sarcomata have the peculiarity of swelling, and becoming slightly painful shortly before and during menstruation. In this disease, the general health is not affected, except that in large ulcerated tumors the patients emaciate, become anæmic, and acquire a suffering look. The course of the disease may vary; there are not a few cases where small sarcomata of the breast, which perhaps came after the first confinement, spontaneously disappeared in the course of time, or else remained for the rest of life without doing any harm; but in most cases these tumors grow gradually, until they are operated for; if this is not done till late, when the tumors have become large, and the women have attained old age, they may become infectious. In young girls and women, when a slowly-growing sarcoma of the mammary gland is extirpated, it does not usually reappear. If, however, the sarcoma first appears between the thirtieth and fortieth years, we have to fear general sarcoma infection, or actual transformation to carcinoma by epithelial proliferation. I consider it advisable, in all cases, to extirpate these mammary sar-

Fig. 147.



From an adeno-sarcoma of the female breast: *a*, dilatation of the excretory ducts; *b*, of the acini, magnified 60 diameters; *c*, a dilated acinus of the mammary gland, with cylindrical epithelium; intermediate substance resembling granulation-tissue, magnified 350 diameters.

comata early, as we never know exactly what their future course will be. The diagnosis is often difficult; small, nodular, lobulated hardenings may occur in the breasts from chronic inflammation, especially during and after lactation, which pass off spontaneously, or under the use of iodine. We often have to decide from the course whether the case is one of chronic inflammation which may subside, or an actual tumor. Even the most accurate anatomical examination is here of no avail, for young sarcoma-tissue cannot be distinguished from inflammatory neoplasia. This is another case where the boundary between chronic inflammatory neoplasiæ and tumors cannot be accurately drawn.

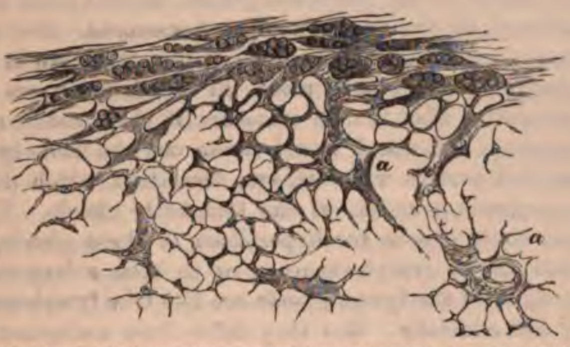
A second organ, in which adeno-sarcoma and adenoma develop, is the salivary gland. The tumors that form here are usually quite firm and elastic, are tolerably movable and grow very slowly; they occur in the parotid more frequently than in the sub-maxillary gland, and very rarely in the sublingual. As seen by the naked eye, the anatomical characteristics vary greatly; the tumor is always distinctly bounded by a capsule, which is very intimately connected with the gland-tissue. The substance of the tumor may be of pulpy, cartilaginous or fibrous consistence, it may be ossified, or calcified; it often contains cysts of briny, gelatinous, or serous fluid. Histological examination of these tumors shows that their softer parts consist of spindle-cells and stellate cells, sometimes with a slight, again, with a large amount of mucous or cartilaginous intercellular substance; there are also newly-formed gland-tubes. In rare cases, the tumor consists almost exclusively of cartilage, but very frequently there is some sarcomatous tissue present. These tumors may develop from the time of puberty to the fortieth year; they grow very slowly and painlessly, and particularly slowly when they do not form till middle age. Although they never retrograde, small tumors (say as large as an egg) of this variety may cease growing late in life. If these tumors be extirpated from young patients, as a rule, they do not return. But later in life they often recur after extirpation, and return so quickly, that they gradually grow deeper in the neck, and finally become inaccessible to the knife; the neighboring lymphatic glands of the neck are infected, and the disease assumes the character of carcinoma; the adeno-sarcoma becomes cancer of the gland. General development of sarcoma scarcely takes place from these tumors. From the course above described, we might form the rule of removing these tumors early in young patients, but in older ones of not being too hasty about extirpation, as rapid recurrence is to be feared, while occasionally the primary tumors grow slowly. Sarcomata of the salivary gland are not frequent. Similar myxo-sarcomata and myxo-chondromata occasionally develop in the oral mucous membrane.

## 9.—LYMPHOMATA.

THESE neoplasiæ are very difficult to define accurately. According to the mode of development we may assume a secondary inflammatory swelling of the lymph-glands from infection, and an idiopathic hyperplasia. In diseases from the most varied causes, the lymphatic glands almost always present a similar appearance; they are enlarged, more succulent, firmer than normal. The microscopic examination of lymphoma shows the following appearances, if made from a hardened, properly-prepared specimen: All the cellular elements are multiplied and enlarged; the lymph-cells in the alveoli, the connective-tissue cells of the trabeculæ, the capsules of the alveoli and the net-work; thus, the structure of the gland is gradually lost entirely; the whole organ becomes a mass of lymph-cells, although a fine net-work is generally preserved, into which the hard connective tissue of the capsule and of the trabeculæ is also transformed, while the blood-vessels are preserved, and their walls greatly thickened (Fig. 148); the cellular infiltration may be so great, that an exact distinction between lymphoma and glio-sarcoma (Fig. 148) may be impossible at some points. Usually there are glands of various sizes, and we find the large ones of the same structure as the smaller. Neither the macroscopic nor microscopic appearances will determine exactly the causes of the hyperplasia, whether it be idiopathic or due to chronic inflammation; we can only say, in general, that glands much enlarged by chronic inflammation more frequently contain abscesses and caseous foci than those which are apparently idiopathic hyperplasia. Perhaps I am too conscientious in using the term "idiopathic disease of the lymphatic glands;" for in many of these cases we can discover no peripheral irritation, although many things speak in favor of the disease of the glands being secondary; it is possible that slight, temporary inflammations have existed, that have excited disease of the glands, and have disappeared before the affection of the glands has shown itself. We formerly spoke of a similar secondary plastic process in the lymphatic glands, after the primary peripheral irritation had ceased, as being a chief symptom of scrofula; hence we might term lymphomata as typical scrofulous tumors (scrofulous sarcoma, *B. von Langenbeck*). Let us study them further, anatomically and clinically.

For a long time the glands preserve their kidney-shape till finally, as they continue to grow, this also is lost, and the adjacent glandular tumors unite to form a lobulated mass. To the naked eye, the extirpated tumors appear roundish, oval, or kidney-shaped; on section, they are of a light, grayish-yellow color, which, on exposure, changes

Fig. 148.



From the cortical layer of a hyperplastic cervical lymphatic gland. Magnified 350 diameters. *a*, section of vessels with thickened walls, brushed-out alcohol preparation.

to a yellowish-red. These tumors are firm and elastic; they are easily diagnosed, from their locality. All lymphatic glands are not equally disposed to this disease; the most frequently affected are the cervical either on one or both sides; more rarely the axillary and inguinal, most rarely the abdominal and bronchial. These tumors are hardly ever congenital, but they may occur from the first to the sixtieth year, although they are most frequent between the eighth and twentieth. Not unfrequently, hyperplasia of the lymphatic glands is multiple; but only one or a few glands in the neck may be affected; if this be the case, the tendency to such neoplasia runs out in the course of time, while the tumors which have grown painlessly, and continued free from pain, have their growth arrested, and may be carried till death. In rare cases, the new formation appears almost at the same time in all the lymphatic glands of one or both sides of the neck, so that the latter is thickened, and the movements of the head are much impeded; if these tumors continue to grow, they finally compress the trachea and cause death by suffocation; but even in these severe cases there is occasionally a spontaneous arrest of the disease, and then even large tumors of this kind may be successfully extirpated; some of these glands, too, are finally destroyed by ulceration and caseous degeneration.

The worst cases are those where the tumors quickly grow to large *medullary tumors* (not unfrequently under the form of fasciculated medullary fungi), and where the neighboring tissue is also changed to lymphoma. Patients with such tumors rarely escape; anæmia comes on, the nutrition is impaired, and hypertrophy of the spleen may appear, and the patient die of excessive anæmia and marasmus.

[These *malignant lymphomata* are to be regarded as true medullary sarcoma of the lymph-glands. But, in regard to the nomenclature of tumors of the lymphatic glands, we find great confusion, chiefly induced by the expression *lymphosarcoma*. For while, by this term, some understand sarcomata, whose structure is like that of lymphatic glands, others mean all primary tumors of these glands, no matter what is their structure, if their course resembles that of sarcoma. Hence I would strike out the name lymphosarcoma, and call tumors with sarcoma formation, which develop in the lymph-glands, sarcoma of the lymphatic glands. The name lymphoma would remain for hyperplasia of these glands, whether due to increase of lymph-elements, or to their enlargement. At first, sarcomata of the lymph-glands are like true lymphomata, anatomically and clinically. But they differ from malignant lymphomata, because the growing tumor quickly bursts the gland-capsule, and attacks the surrounding parts, becoming adherent to them, and not unfrequently causing ulceration and extensive destruction of the skin. They are most prone to recurrence, and belong to the most dangerous form of tumors. Quite lately I have seen several cases where metastatic tumors, with alveolar, round-celled sarcoma structure, were found in the lungs and spleen.

Besides these malignant sarcomata of the lymph-glands, there are *malignant lymphomata* (*Billroth*), which, in another way, are scarcely less dangerous, though not possessing all the clinical peculiarities of malignant tumors—among others, unlimited tendency to spread, and consequent destruction of organs. These tumors are true hyperplasias of the lymph-glands, the proliferation of the elements attacking a group of glands at once, but remaining limited to the glands, so that the capsule is not ruptured or the neighboring parts attacked. The glands first attacked grow to a tumor composed of several nodules, which is at first painless. Then an adjacent group is attacked, and the disease spreads to the other side of the body, and, finally, to the mediastinal and retroperitoneal glands. Occasionally, by this time, the patient's general condition has suffered; the skin is pale, the body emaciated, there is œdema of the limbs; later, they cough, have hectic, and severe pain in some of the tumors, and finally die from general exhaustion, bloody diarrhœa, cough, with purulent expectoration, etc. But this result is not the rule; some patients, especially old, strong men, have malignant lymphoma, and, in spite of enormous development of the tumors, maintain good general condition. They may suffer solely from displacement and compression of the larynx and trachea, and it is astonishing what patients can stand in this way, when the compression comes on gradually. Usually these patients die suddenly from suffocation, often after severe exertion, like goitre patients.

On autopsy, we usually find several groups of glands, but sometimes only those of one side of the neck, immensely enlarged; similar tumors are found in the mediastinum and abdomen. But sometimes true lymphoma nodules are found in the internal organs, in the lungs, spleen, liver, kidneys, and even in the spongy bones, which may be regarded as metastases. These tumors are classed as hard or soft, according to their consistence; the former are a higher form of development than the latter, but the two are distinctly characterized from the start: the soft have a gray-reddish medullary appearance, the hard are pure white or grayish-white, like fibroma. The same difference is found in the metastatic tumors.

In the soft as in the hard lymphomata, the lymph-vessels are permeable; in the former they are even easy to inject. The general structure of the gland is preserved; in the channel, large, multinucleated, often giant cells, are found. In the hard forms, the reticulum particularly is thickened and changed to sclerosed connective tissue; in the soft, there is mostly new formation of lymph-cells and dilatation of the framework. The disease usually begins in the cervical glands of one side, and sometimes remains limited there. Not unfrequently the glands of the throat and of the tonsils are attacked, and early cause difficulties of swallowing and respiration. More rarely the affection begins in the axilla; least often in the groin. Sometimes a slight irritation is the exciting cause, as, in one patient, a bee-sting on the hand started an acute lymphangitis and adenitis of the axillary glands; this latter did not subside, but grew like a tumor. In other cases a slight glandular swelling is caused by an irritation continued several months; suddenly the glands begin to grow rapidly, and neighboring groups are attacked. Usually there is no perceptible cause. Scrofulous persons, in whom chronic glandular hyperplasia is so common, are not disposed to malignant lymphoma; hence, we may decide against the disease being of this nature, if we find the marks of scrofula. No age is exempt—it occurs in children of a few months or in persons sixty years old; but healthy-looking persons, in early puberty, seem most prone. Some cases had been preceded by intermittent fever that had left no sequelæ.

The great resemblance of malignant lymphoma to so-called leucæmia lymphatica, in which also there are tumors of the spleen and other organs, has often been observed, and the affection has been called *pseudoleucæmia* (*Trousseau*). But the two diseases are distinct in the structure of the hyperplastic glands. A more important point is the absence, in malignant lymphoma, of the characteristic of leucæmia; i. e., increase of the white corpuscles. In the last stages of pseudoleucæmia, as in marasmus from any cause, the proportion of colorless corpuscles is increased, but never so markedly as in leucæmia proper. Moreover, patients with the latter disease are ailing from its commencement or even before; while those with malignant lymphoma, even when the tumors are large, are usually strong and feel well. The disease is local. When, in some cases, there is marked leucocythæmia, it is probably due to special pathological conditions; for instance, from the growth of the tumor into the axillary vein, etc. In true leucæmia, also, the medulla of the bones is implicated, while in malignant lymphoma it is not.

There are other hyperplasias of the gland that can neither be termed chronic scrofulous lymphadenitis nor secondary glandular swellings. Of course, we do not mean those glandular enlargements near a neoplasia, or their symptomatic enlargements. These tumors have the formation of hypertrophied glands, but no suppurative or caseous degeneration; they are movable, painless, hard, isolated; they usually grow in the neck, perhaps to the size of a hen's egg—so that we might suspect malignant lymphoma; but their growth ceases suddenly, and other groups do not become diseased, and the tumors remain stationary; if removed, they do not return.

From the above, you see the prognosis varies. In the commencement you can never tell what will become of a lymphatic-gland tumor. Rapid growth and adhesion to the surrounding parts and skin indicates development of the most malignant medullary sarcoma, and a very active course. If several groups of glands enlarge, even if there is malignant lymphoma, a fatal result is certain; but the duration of the disease depends on the rapidity of the growth, and the complications caused by the location of the tumor and the extension to internal organs. The softer forms seem to grow more rapidly, and to cause more metastases; young persons and those in whom the disease is extensive at the start die sooner than persons beyond forty years. Cases with rapid growth usually die within two years; others, probably the majority, last four or five years. Malignant lymphomata furnish a large proportion of mediastinal tumors, which cause the most varied symptoms, and always prove fatal in three or four years.

Of all the lymphomata, the only ones free from danger are the last-mentioned, simple hyperplasiæ, enlargement of single glands not going beyond a certain size, which may remain stationary for life, and do not suppurate or become caseous. This affection begins at the commencement of puberty or sooner; after the thirtieth year all such tumors are suspicious.

The *treatment* of the above diseases at first, until we are sure it is not a scrofulous swelling of the gland, is internal—such as cod-liver oil, brine-baths, and preparations of iodine, unless it is contra-indicated; with commencing anæmia, iron is indicated alone, or combined with iodine. In rare cases under this treatment, recent lymph-tumors recede. *Lücke* made parenchymatous injections of tincture of iodine with good effect; according to my experience, the tumors are thus diminished, but chiefly by the formation of abscesses which are opened, and the resulting cicatrices contract. I have rarely seen disappearance of the tumor by absorption without suppuration. The same is true of the constant current. Of external remedies, mercury has almost no effect; painting with tincture of iodine is most efficacious; it sometimes causes periadenitic swelling, adhesion of the glands to each other and to the skin, so that subsequent extirpation may be difficult. *Baum* has tried continued compression by a special apparatus; this requires time, and is rarely effectual. If it is a case of simple, long-stationary swelling of the lymphatic glands, it is best to extirpate them all, or at least the larger ones. In *malignant lymphoma*, in some cases *Fowler's* solution of arsenic has proved effectual, by parenchymatous injection and internally, in increasing doses till symptoms of poisoning. Under this treatment, large tumors, even in the chest and abdomen, receded in a few weeks, usually with severe fever. We begin the treatment with ten drops of *Fowler's* solution internally, and inject two drops into the tumor every day; every third day the internal dose should be increased by two drops; the injections should be given in different glands, and gradually increased to four or six drops. If symptoms of poisoning occur, the doses should be reduced, but never suddenly withdrawn. I have seen many astonishing results from arsenic treatment, even in cases of recurrence, but no permanent cures. In sarcoma of the lymphatic glands arsenic is much less useful; but in simple glandular hyperplasia injections are beneficial.

In the "Wiener medicinische Wochenschrift," 1871, No. 14, *Billroth* published about the first successful case of treatment by arsenic. A woman forty years old was sick for ten months with the glands of the axilla, neck, and groin as large as an egg or a fist. Quinine was ineffectual, but after using *Fowler's* solution of arsenic for a fortnight the glands had begun to diminish in size, and at the end of two months, when dismissed from treatment, there remained only a gland as large as a filbert on the neck.

*Winiwarter* reports five other cases, in three of which there was no benefit; in one the glands disappeared rapidly, but the patient died from inflammation induced by the removal of a small gland from the neck for the purpose of verifying the diagnosis; one case was still under treatment.

In the commencement of the treatment the remedy was well borne; the first sign of its action on the glands was that they became softer, and after eight or ten days were almost always painful. Subsequently, when the treatment was successful, the glands rapidly became smaller, and at the same time harder and gradually less sensitive. In one case there were inflammation and suppuration of the tumors.

Operating is of no use in malignant lymphoma. Recurrence is usually so rapid, that operations cannot keep pace with it, and, after numerous operations, the patients die sooner than if left alone; it would only be advisable where disease of a few glands had run its course. But we often have to make partial extirpation, on account of difficulty of respiration due to tumors in the tonsils and neck near the trachea.

In sarcoma of the lymph-glands, in the early stages, we may operate, and effect at least temporary benefit.]

The operation itself will be well borne in cases where the glands may be isolated, and still preserve their capsules. I have extirpated (or rather dug out with my finger) twenty or more isolated glands from the neck of the same patient without subsequent recurrence; but when the glands unite to one mass, and are soft, it is on the one hand a sign of rapid growth, and local recurrence may be certainly expected; on the other hand, it will greatly increase the difficulty of operation. Sometimes lymphomata, developing deep in the neck in young, otherwise healthy persons, grow behind the jaw into the throat and implicate the tonsils and pharynx; they usually soon prove fatal; the operations that might relieve them are so dangerous that they rarely prolong life.

Of the other glands, which, according to recent observations, are to be classed in the lymphatic-gland system, the *tonsils* alone are subject to hyperplastic disease; but this hypertrophy of the tonsils which is common, and in children and young persons is quite frequent, more resembles chronic inflammatory secondary swelling of the lymphatic glands; it is usually the result of chronic catarrh of the pharynx, while the reverse is often falsely considered to be the case, namely, that the hypertrophied tonsils are the cause of the pharyngeal catarrh; hence, in such cases, extirpation does nothing for the chief trouble, the frequent inflammations of the throat.

Hypertrophy of the *thymus gland* does occur, but is rare. The analogous diseases of Peyer's glands and the spleen have no special interest in surgery.

Lymphoma also occurs in tissues which do not belong to the lymphatic glands. I class as lymphomata all those medullary tumors, usually soft, in which, by hardening and preparation, we may see a net-work analogous to that of the lymphatic glands. In this sense, I have seen lymphomata of the upper jaw, scapula, cellular tissue, eye, etc.; tumors whose structure frequently can only be imperfectly distinguished from granulation sarcoma (especially from *Virchow's* gliosarcoma), and which form their ordinary medullary consistency, are briefly called "medullary fungi." According to my experience, the mixture of the above forms has no special prognostic significance, as these tumors are alike malignant and infectious; but the importance of the most accurate examination of these tumors should not on this account be limited or undervalued; during the last ten years we have learned interesting and important clinical differences for the more accurate distinction between sarcoma and carcinoma. Ten years ago we could not have spoken as decidedly about sarcoma and lymphoma as we now may. What we now include under "lymphomata" were formerly treated of partly under glandular hyperplasiæ, partly as sarcomata, partly as medullary fungi.

## LECTURE XLIX.

10. *Papillomata.*—11. *Adenomata.*—12. *Cysts and Cystomata.*—Follicular Cysts of the Skin and Mucous Membranes.—Neoplastic Cysts.—Cysts of the Thyroid Gland.—Ovarian Cysts.—Blood-Cysts.

### 10. PAPILLOMATA—PAPILLARY HYPERTROPHY.

HITHERTO we have spoken exclusively of new formations from the series of connective-tissue substances, muscles and nerves. We now pass to the neoplasiæ of true epithelium, derived from the upper and lower germ-layer of the embryo.

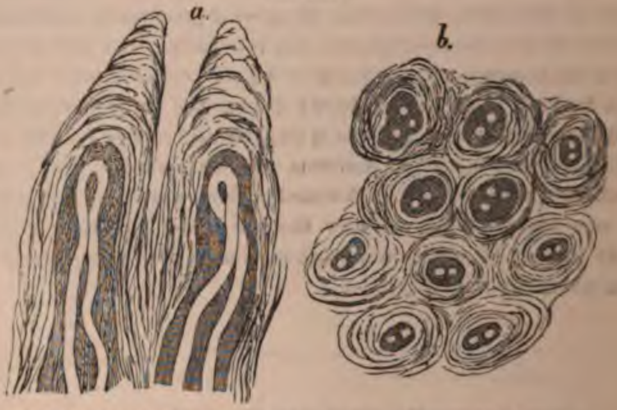
The epitheliums form a great part of two normal tissues, namely, of the papillæ (tufts, intestinal villi), and of the glands; the former are wavy or finger-like elevations, the latter pouched or cylindrical sinkings in of the membranes, which the epithelial covering accurately follows. Both give the physiological paradigms for certain forms of tumors, of which we shall mention the purely hyperplastic forms of the first series, *papilloma*, and those of the second series, *adenoma*

Both are accompanied by corresponding connective-tissue and vascular neoplasia.

Horny papillomata come exclusively in the cutis, rarely in the walls of sebaceous cysts. We may distinguish two chief forms:

(*a.*) *Warts.* Anatomically these consist of an excessive growth in length and thickness of the papillæ. The epidermis on these abnormally large papillæ hornifies in the form of small rods, of which every wart is composed, as you may readily see with the naked eye (Fig. 149). These warts which, without any known cause, appear especially often on the hands in great numbers, are rarely larger than lentils or peas.

FIG. 149.



Wart: *a*, longitudinal section; *b*, cross section. Magnified 20 diameters.

(*b.*) *Horny excrescences* are to some extent large warts; the epidermis of the enlarged papillæ adheres to a firm substance, which increases enormously, so that the horn, whether it be straight or twisted, may grow to three or four inches or more. Although externally these horns greatly resemble those of some animals, their anatomical structure is different, for the latter always have a basis of bone. Horny excrescences are of a dirty-brown color; they occur chiefly on the face and scalp, but may also come on the penis and other parts of the body, and occasionally they grow from atheroma-cysts.

The development of warts and horny excrescences is evidently due to a general tendency of the skin that way. This is chiefly evident from the fact that as many as twenty or thirty warts often occur on the hands, especially of children shortly before puberty. Irritating external influences, affecting the hands particularly, apparently combine with the fact that the epidermis on the hands is normally very thick

The tendency to horny excrescence, rare as it is, rather belongs to advanced age, just as most of the other epidermoid neoplasiæ, of which we shall hereafter speak. Anatomically, *hystricismus* would also belong to the above forms of horny growths. *Hystricismus*, or porcupine-disease of the skin, is a peculiar variety of papillary hypertrophy, with hornifying of the epidermis of such a nature that porcupine-like formations develop on the cutis. Like *ichthyosis* (a scaly thickening of the epidermis over the whole body), this affection is mostly congenital; but I have seen analogous formations in some forms of elephantiasis nostras.

The predisposition to warts is entirely devoid of danger, and in many cases ceases spontaneously. Popularly, warts are considered contagious, possibly not altogether without reason. I saw a case where an ordinary wart formed on the side of a toe, and, on the part of the neighboring toe lying in contact with it, another wart formed. Horny excrescences are more important; although they occasionally break and fall off spontaneously, they grow again if they are not operated upon; indeed, in some cases epithelial cancer forms at the point where a horny excrescence was located.

In most cases warts may be left to themselves. As in all diseases that recover spontaneously in the course of time, there are numerous popular remedies: old women regard the placing of a hand covered with warts on the hand of a corpse, or rubbing it with various leaves and weeds, as sovereign remedies. If you wish to get rid of certain large warts that are peculiarly annoying to their owners, it may best be done by caustics. For this purpose I use fuming nitric acid, applying it to the wart and the next day cutting off the cauterized portion till a drop of blood flows, then repeating the cauterization. This should be continued till the wart has entirely disappeared.

Horny excrescences can only be cured radically by cutting out the piece of skin on which they are located.

By *soft, sarcomatous papillomata,* we mean those neoplasiæ that have the form of papillæ, consist of soft connective or sarcomatous tissue, and are covered by an epithelial coating analogous to that of the matrix.

Sarcomatous papillæ (soft warts) occur rarely on the cutis, but occasionally appear congenitively on one side of the face as cock's-comb-like proliferations. The broad and also the pointed condylomata on the mucous membranes are products of syphilis and of the specific irritating pus of gonorrhœa; we do not class them among tumors.

Sarcomatous papillomata develop much more frequently on the mucous membranes, especially on the portio vaginalis, more rarely in the rectal and nasal mucous membrane. According to the surgical nomenclature hitherto in use, they come in the category of mucous polypi. They are often complicated tumors, in which proliferation and ectasia of the glands, formation of sarcomatous intermediate substance, and papilloma, all go together. They are mostly pedunculated tumors; occasionally a large surface of mucous membrane becomes diseased at the same time.

These papillomata are rarely infectious, but they sometimes recur after extirpation. The extensive papillomata that occasionally occur in the larynx in children are perhaps always of syphilitic origin.

I formerly called tumors with papillary formation, which developed from vitreous mucous tissue, *cylindromata;* but this formation is not so characteristic as I formerly supposed; it occurs both in sarcomatous and carcinomatous tumors. Fibromatous and sarcomatous papillæ may develop on the inner surface of cysts.

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## 11. ADENOMATA—PARTIAL GLANDULAR HYPERTROPHY.

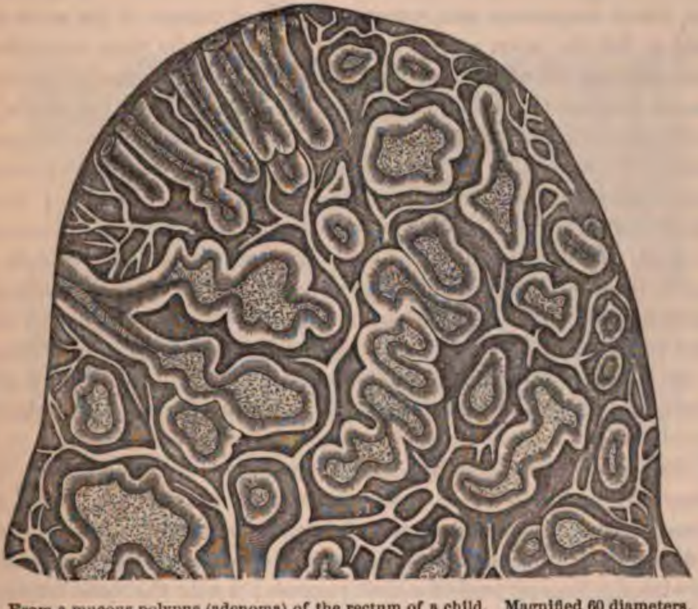
New formation of genuine, regularly-developed glands or parts of glands is not frequent, although we shall hereafter learn that, in cancer, incomplete development of glands is one of the most common forms of neoplasia.

Although sarcoma of the mamma was often spoken of as partial hyperplasia of the gland, because glands were found in it, of late it has appeared doubtful whether gland-acini were really developed in the tumors formerly described as adenosarcoma (page 731); from my own observations, I must consider true adenoma of the breast as very rare; I have only seen it once, it was then in a tubular form. *Förster* and others, however, describe acinous adenoma of the mamma; on account of this rarity, not much can be said about the prognosis of these tumors, which usually remain small. They are generally considered as entirely benignant; but, on anatomical grounds, it seems to me probable that they cannot differ so much in prognosis from carcinoma.

So far as my investigations go, the so-called *hypertrophy of the prostate* is never accompanied by development of adenoma, but only by ectasia of the acini and epithelial hyperplasia; the frequently-observed enlargement of this gland depends essentially on diffuse or nodular myoma (page 704).

The glands of the skin and some mucous membranes may also give rise to development of adenoma and adenosarcoma; it is said that tumors of the skin, which are to be regarded as pure adenomata, may result from the glandular epithelium, analogous to the gland-development in the fœtus. *Verneuil* first described an adenoma of the sweat-glands. I have never observed such tumors, but do not doubt their existence, since *Rindfleisch* has demonstrated to me an adenoma of this variety. Those glandular formations that occur in the mucous membrane of the nose, rectum, and uterus, and which are embedded in a gelatinous, œdematous connective tissue, more rarely in some other form of sarcoma-tissue, are more frequent.

FIG. 150.



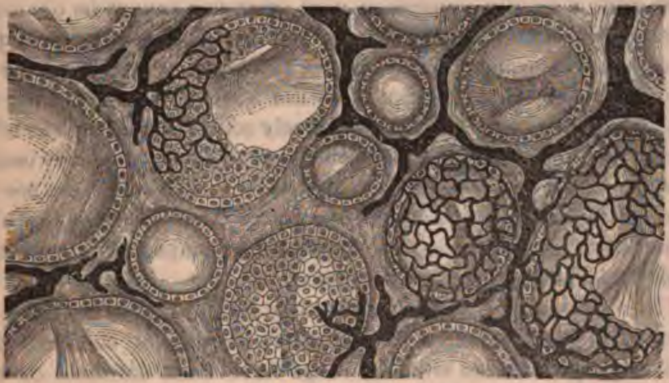
From a mucous polypus (adenoma) of the rectum of a child. Magnified 60 diameters.

Tumors are thus developed which, in general terms, are called *mucous polypi:* sometimes they are in broad folds, sometimes nodular pedunculated tumors; they have the color and consistence of the mucous membrane whence they spring, are also covered with its epithelium, except only the soft polypi of the external auditory meatus; strange to say, these are sometimes covered with ciliated epithelium. All of these mucous polypi do not contain glands; they are usually absent from the aural polypi and the small, leaf-like proliferations of the female urethra, the so-called urethral caruncles. The latter neoplasiæ consist solely of œdematous and gelatinous connective tissue, with an epithelial covering. Most mucous polypi of the nares, large intestine, and especially of the rectum, consist to a great extent of elevated and also newly-formed glands of the mucous membrane, whose closed ends sometimes dilate to mucous cysts. Hence, in the anatomical system, according to the glands they contain, mucous polypi may be classed among pure adenoma (as rectal mucous polypi in children), among adeno-sarcomata (many nasal mucous polypi), among œdematous fibromata, or, lastly, among the myxosarcomata. The predisposition to mucous polypi reaches from infancy to the fiftieth year. In children the disease is limited to the rectum and large intestine, where sometimes one, sometimes several tumors of the same sort develop, but the latter occurs even oftener in adults than in children. From puberty till about the thirtieth year, it affects chiefly the nasal mucous membrane; sometimes giving rise to single polypi, again, to proliferations in both sides of the nose; the latter is the more frequent. Toward the thirtieth year, mucous polypi of the uterus occur; under some circumstances they may change to cancer. In all of these polypi there is a great tendency to recurrence, especially in those of the nose, which often do not cease growing till they have been removed three or four times. Generally, in the course of years, the disposition to these new formations ceases spontaneously, and they cease to recur, or the smaller ones even cease to grow, as, for instance, in the uterus. Microscopic examination of these tumors may give some clew to the prognosis, inasmuch as those tumors which consist entirely of œdematous connective tissue have far less tendency to recur than those which consist of tissue analogous to inflammatory new formation; lastly, in some cases anatomical examination alone can prevent mistaking them for epithelial carcinoma.

Mucous polypi of the nose are most readily removed by tearing them out with the forceps made for that purpose; we do the same for those of the external auditory meatus [the latter may be most effectually cured by free applications of liquor ferri persulphatis]; those of the uterus and rectum we may cut off at the base with scissors; if we fear hæmorrhage, we may previously apply a ligature, or employ the écraseur.

Of the glands without excretory ducts we shall here consider only the *thyroid*, as it is a true epithelial gland; adenoma of the ovary so often becomes cystoid in form, that it may be more suitably treated of in the next section. Tumors of the thyroid gland have long been called *goitre*, *struma* (in the middle ages "strumous" indicated what we at present call "scrofulous"). Considering the anatomical relation of these tumors to the gland, we find that there are diffuse swellings of the gland, affecting one or both lobes, and others that are distinctly bounded in the gland, the latter remaining normal or but slightly hypertrophic. If we exclude simple cysts of the thyroid, so-called struma cystica, most other forms of goitre are pure adenoma or cysto-adenoma. If the tissue of these tumors, which may vary greatly in consistence, be not metamorphosed by secondary changes, on section it appears to the naked eye almost the same as the cut surface of a normal thyroid gland. Microscopically also it is very much the same; almost all thyroid tumors on microscopic examination show a large amount of connective-tissue capsules, which contain a clear gelatinous substance filled with more or less round pale cells (Fig. 151). The size of these varies greatly, the youngest, which as yet contain no gelatinous substance, but only cells, being analogous to the fœtal thyroid vesicles, while the larger are six or ten times this size. One of the most frequent changes in goitre-tumors is the formation of cysts, which come from a number of the dilating gland-vesicles uniting, and their thick gelatinous contents becoming fluid. But, besides this formation of cysts in goitres, there are other just as frequent changes that occur almost regularly if the goitre exists a long time: these are extravasations of blood, which are mostly reabsorbed, but leave more or less pigmentation. Caseous and fatty degeneration is also frequent in old goitres; lastly, calcareous degeneration often occurs, so that by these secondary changes the original picture of the tumor may be much altered. Goitrous tumors, which may lie in the middle of the

FIG. 151.



From an ordinary firm tumor of the thyroid—adenoma of the thyroid; partial injection. Magnified 100 diameters.

neck or to both sides, in numbers or solitary, may attain a considerable size, compress the trachea, and cause suffocation. Much more rarely the regular double-sided hypertrophy of the thyroid attains a dangerous size. Goitre is chiefly remarkable for its endemic occurrence; it is found mostly in mountaineers: it is seen in the Hartz, Thuringian, Silesian, and Bohemian mountains, and in the Alps, although not equally frequent in all parts. Some valleys of Switzerland and of the Austrian Alps are entirely free from it. It has been ascribed to the most different causes, especially to the water and soil, without any definite scientific reason having been found by accurate investigations. Undoubtedly, climatical and geological conditions have much to do with this disease. Complete similarity in the constitution (probably often hereditary) of goitrous patients can hardly be proved; a certain connection with cretinism cannot be denied, inasmuch as most cretins have goitre; but the disease is more frequent in persons with well-developed bones and brain. Goitre may be congenital in some rare cases, but does not usually increase till the commencement of puberty; the growth rarely continues beyond the fiftieth year; goitres which have continued harmless till then, usually cease to grow, and subsequently cause no trouble; to this rule there are only a few exceptions, where cancerous goitre develops from the above hyperplastic form, infecting the neighboring lymphatic glands; these almost always prove fatal by suffocation.

[But cases where death is due solely to the goitre are more frequent than was formerly supposed : it may occur suddenly from suffocation ; when the tracheal rings have been softened or worn away by the tumor, so that there remains only a membranous tube, rapidly turning the head to one side may close the tube entirely and cut off respiration. *Rose* first called attention to this danger, and explained the cases of sudden death.]

It is scarcely necessary to consider *struma aneurysmatica* as a peculiar variety, as it is merely a goitre accompanied by great dilatation of the afferent arteries. Preparations of iodine are usually employed against this disease ; they are only efficacious, however, at the commencement. [They are used internally (moderate doses of iodide of potash) as well as externally by painting and as parenchymatous injections into the tumor ; these injections of tincture of iodine are highly recommended by *Lücke* and *Schwalbe ;* diluted alcohol may be used the same way, also solution of ergotine and iodoform emulsion (℞ Iodoformi, glycerini, aquæ destill., āā partes equal. ; mucilago gum. arab., qu. sat. ut fiat emulsio) ; these substances seem to have about the same effect.]

In the first cases where I used these parenchymatous injections of tincture of iodine, they had no effect; one case where I injected alcohol proved fatal from suppuration of the goitre and septicæmia. Lately in some cases I have obtained considerable diminution of the goitre by persistent injection of iodine; twice a week I inject one gramme of pure tincture of iodine; this must be continued several months. Under this treatment some patients emaciated greatly, so that I would not recommend it in feeble or tuberculous patients. Since the above-mentioned unfortunate case I have not tried alcohol injections. *Störk* also has informed me that alcohol injections sometimes excite considerable inflammatory reaction, while after injections of iodine there is merely a temporary swelling and pain; it is prudent at first to inject a third then a half syringeful, to test the individual susceptibility of the patient.

[It is hardly necessary to tell you that these injections do no good in cancerous glands. Not unfrequently, as a gland has diminished in size, hectic fever, emaciation, loss of appetite, anæmia, and debility, have came on; some surgeons, perhaps unjustly, regard these as signs of iodine-poisoning. In two cases I have seen this condition where no iodine has been used for months; it is probably due to absorption of the diseased gland-tissue, as occurs in malignant lymphoma under the arsenic-treatment; in feeble or tuberculous patients it may prove fatal. These possibly dangerous results of medical treatment must be duly considered; their possibility is some reason for preferring operation.

Extirpation of hypertrophied thyroid glands has really only become a frequent operation in the last ten years, since we have learned not only how to do it, but also how to secure the wound against reaction by *Lister's* dressing. Formerly only those tumors were removed which from their size or location endangered life; now the operation is more freely resorted to, as we have found the dangers less and treatment by medicines more tedious and uncertain.

The best chances are offered by movable goitres in the median line of the neck in young persons; while even small ones deeply embedded in the lateral lobes, or with part of their periphery behind the sternum or trachea, are not to be removed without difficulty and danger. Even the slightest operation of this kind must be done most carefully, and with skilled assistants, unless the case is very urgent; if there is danger of suffocation, of course the tumor must be removed or the trachea opened under any circumstances. Of late I have removed many hypertrophied thyroids and some goitres with the best results, healing usually taking place with no complications and no deaths. The most important point in the operation is arresting the hæmorrhage: you should not make a cut till by pressure-needles or forceps you have rendered it safe to divide the parts. While the vessels entering or leaving the tumor have thus been secured at the periphery, the tumor is loosened from its attachments and removed; do not tear off adhesions without ligating them; thick veins may look like connective tissue, but bleed furiously if divided. The case is different in removing single encapsulated goitre nodules from the thyroid gland; then by the fingers or a director the nodules may be detached and turned out except the pedicle, a firm cord containing the vessel which is to be ligated. You should never trust to ligating the vessels separately after their division; too much blood would be lost, and it would take too long. After the operation apply an antiseptic dressing, previously arranging the drainage-tubes and sewing up the wound to avoid a scar as much as possible.]

## 12. CYSTS AND CYSTOMATA—CYSTIC TUMORS.

A tumor formed by a sac filled with fluid or pulp is called a cyst or cystic tumor. It may develop from a sac already existing (cyst), or it may develop entirely new (cystoma). If the tumor be formed of a convolution of very many such cystic tumors, it is called a "composite cyst or cystoma." If in one of the tumors already described, or in carcinoma, we find cysts also forming an essential part of the tumor, we give them names like *cysto-fibroma*, *cysto-sarcoma*, *cysto-chondroma*, *cysto-carcinoma*, etc.

*Virchow* classes among tumors encapsulated extravasations (as hæmatoma), dropsical effusions, retention-cysts (as hydrops vesicæ felleæ), and follicular cysts (as in glands of skin).

Among the glands of the cutis, cysts develop from the sebaceous alone; I do not know that cysts of the perspiratory glands have ever been described. The reasons for secretion collecting in the sebaceous glands are: (*a*) its becoming inspissated; (*b*) closure of the excretory duct. If from either of these causes the secretion be retained and collect in the gland, the pouched secreting surface becomes expanded to a simple sphere; the collected secretion exercises a mechanical irritation on the surrounding connective tissue, which consequently becomes thickened and surrounds the secretion like a vesicle. If the sac, not yet grown large, can be evacuated by strong pressure, the small open cyst is called a *comedo*, or "maggot." If, from any irritative inflammatory process, the excretory duct of a sebaceous gland be closed, there may be atrophy of the gland, as after a burn with very superficial destruction of the skin; but in other cases the secretion of the gland continues, and it distends slowly to a large sac. Such cysts, filled with fatty pulp and epidermis, are called pap-bags (*grützbeutel*), atheromata. On microscopic examination we find the pulp to consist of fat-drops, fat-crystals, especially cholestearine, epidermis-cells, and small plates. It has very varied color and consistence; most atheromata on the scalp, which develop at advanced age, contain a dirty-grayish brown, badly-smelling, pulpy, pasty, sticky substance. Other tumors of this sort, especially those that are congenital, on the forehead, temples, or face, are filled with a milky or light-yellow pulp, which, under the microscope, shows little besides epidermis-scales and crystals of cholestearine. This form of atheroma is called "cholesteatoma." The sacs of these cysts are usually thin, and are composed of connective tissue; their inner surface is usually distinctly bounded by rete Malpighii, and is wavy, or elevated into papillæ. I have found no other resemblance to cutis in these sacs, but others have found hairs and sweat-glands in them. The contents of these cysts sometimes become calcareous. Atheroma may rupture as a result of injury, or, very rarely, spontaneously; the pulp is evacuated, the edges of the opening are everted, and the inner surface of the sac becomes a bad-looking, ulcerated surface; except on the head and face, where they are frequent, these tumors rarely occur.

In the neck, salivary ducts (closed internally and externally, but open in the middle, which are lined with epidermis) may, in the course of years, become large cholesteatomata by the deposit of epidermis. These show themselves in the mouth (as ranula), or externally on the neck above and behind the thyroid.

In the mucous membranes, also, inspissation of the glandular mucus and consequent hinderance to its evacuation, may cause development of mucous cysts; but probably the more frequent cause of retention-cysts here is closure of the excretory duct. The secretion in these glands is usually a tenacious, often thick mucus, of a honey-color (meliceris), reddish yellow, or even chocolate-brown. On microscopical examination of the contents of the cyst, we find numerous large, pale, round cells, often containing fat-globules, in homogeneous mucus, also cholesterine crystals, often in large quantities. In the nasal mucous membrane these cysts are rare, but they occur in nasal mucous polypi, often to such an extent as to give them the name of cystic polypi. *Luschka* often found small cysts in the mucous membrane of the antrum Highmori. In the oral mucous membrane they occur chiefly on the inside of the lips, more rarely on the cheeks; they are an ordinary occurrence in the uterine mucous membrane and in uterine polypi. In the rectal mucous membrane, on the contrary, mucous cysts do not occur, and they are very rare in the mucous membranes deep in the body.

*Neoplastic cysts.* These result mostly from softening of tissue previously diseased by cell-infiltration, or of firm tumor-substance. As soon as the new formation has separated into sac and fluid contents, in some cases a secretion from the inner wall of the sac begins, so that the softening cyst becomes a secretion or exudation cyst, and thus grows. Any tissue rich in cells may be transformed into a cyst by mucous metamorphosis of the protoplasm, or, as others express it, by separation of the mucous substance through cells, without any connection with development of mucous glands. In the fœtus, we know there is a development of cavities (i. e., the joints) by mucous softening of the cartilage-tissue. In cartilage-tissue there is often a mucous softening of certain parts, by which chondromata with mucous cysts are developed. In the same way it is not uncommon for parts to become fluid and encapsulated; the same thing occurs in sarcoma, especially in giant-celled sarcoma. The often slit-shaped, smooth-walled cysts, with serous or sero-mucous contents which occur in uterine myomata, are possibly enormously dilated lymph-spaces. Bone-cysts always originate by softening; the often glistening smooth membrane lining such cysts may in the course of time actually secrete.

While the above varieties of neoplastic cysts have no relation to gland new formations, those we are now about to mention develop from adenoma. The cysts of the thyroid, cystic goitre, already mentioned (page 749), have a somewhat uncertain position in this series; uncertain because they are not due to newly-formed gland follicles or ducts, but to collection of mucous secretion in one of the thyroid vesicles. If we term the contents of these cysts secretion, as we might do for some reasons, we must class these cysts as retention-cysts. But, as it might be urged on the other hand that it would be questionable to speak of a secretion of the thyroid gland, as some state that normally the contents of the thyroid vesicles consist solely of cells, we may also consider the cysts resulting from softening of the contents of the vesicles as newly formed. Whichever view we take, it is certain that the cysts of the thyroid may be solitary, and may attain great size. Moreover, in almost every large, and in some small, otherwise firm goitres, one or more cysts occur; they usually have very smooth walls. The large, isolated cysts of this variety, particularly, give the impression that they are chiefly secretion-cysts, while other similar cavities in other parts of large goitres, by their softened, ragged walls, give the impression of being softening cysts. In the thyroid gland the process of softening usually terminates in the formation of a mucous fluid·

but there are other cysts in these glands that contain a gray, friable pulp, which looks like that from sebaceous glands, but differs essentially from it because it contains only the detritus of thyroid tissue; I have never seen genuine atheroma-pulp in thyroid cysts.

Among the complicated cystic tumors are the cysto-sarcomata of the breast, of which we have already spoken (page 731), cystomata of the ovary and testicle, cysto-adenoma, cysto-sarcoma, and cysto-carcinoma. According to recent investigations, in the great majority of these cases there is a new development of gland follicles or ducts, from which terminal swellings become choked off, as results normally in the development of thyroid or ovarian follicles. A mucous wine-yellow, brownish-red, or dark-brown fluid is secreted in these newly-formed follicles (perhaps also in the normal ovarian follicles); this gradually distends the follicle, which was at first microscopic. Sometimes immense ovarian tumors (distending the abdomen more than it is in the ninth month of pregnancy) may develop from such a follicle, or from the confluence of several of them to a common cavity. In other cases, hundreds or thousands of such follicles develop, forming the multilocular cystic tumors of the ovary. The latter process also occurs in the testicle, although more rarely than in the ovary. In both of these organs, as in the mamma and thyroid, the contents are mucous as a rule; but, in the neoplastic follicular cysts of the ovary and testicles, there are occasionally secretion of fat and extensive production of epidermis; these may remain as epithelial or epidermis pearls (cholesteatoma pearls, page 753), as big as a millet-seed or a pea, as I have seen them in tumors of the testicle, or form large cysts containing fat-pulp. The walls of these cysts, which are found the size of a child's head or larger, in the ovaries of old women, are usually more highly organized than those of cutis atheroma; large quantities of hair, sebaceous glands, sweat-glands, papillæ, even warty growths, are not unfrequently found in them. Indeed, plates of cartilage and bone, with teeth of varied form, have been found in these cysts, so as to render it probable that they were aborted fœtuses from an incomplete ovarian pregnancy.

Besides occurring at the above positions, composite cysts are occasionally congenital about the sacrum; they often contain ciliated epithelium, and, besides other tissues, they sometimes have glandular, follicular formations. The tissues in these congenital tumores coccygei vary from the relatively simple forms of cysto-sarcoma to the fœtus *in fœtu*, and cannot here be further entered into without going into details and fine discussion.

I must lastly mention cysts containing perfectly fluid venous blood, and having smooth walls, which are here and there mentioned in literature. Some of them refill rapidly, others more slowly, after puncture; such cysts have been observed in the axilla, on the thorax and neck. Excluding those cases where effusions of blood have given a dark blood-color to the mucous of serous contents of a cyst, and considering only those in which there is blood alone in the cysts, they could scarcely have been any thing but large sacs on the veins or cavernous-venous tumors whose framework had been entirely atrophied. All the cases of this kind so far reported have been cured by puncture and injection with iodine, so that nothing can be said of the pathological anatomy.

The *diagnosis* of cystic tumor is easy; if it can be certainly palpated, the fluctuation will be felt; deeply-seated cysts are often difficult to recognize. They may be mistaken for other encapsulated fluids; an exploratory puncture with a very fine trocar is admissible to confirm the diagnosis, if this be necessary to determine the treatment. There are various things for which a cyst may be mistaken; e. g., cold abscesses are also painless, occasionally very slowly enlarging, fluctuating tumors; also cystic parasites, of which two varieties occur in the outer parts of the body, especially in the subcutaneous tissue; *cysticercus cellulosæ* and *echinococcus hominis*, although rare, do occur in the cellular tissue (and still more rarely in bone); the former is a small, the latter a large vesicle, which may contain many smaller ones; the vesicle of which the animal consists always has a neoplastic sac around it; as may be readily seen, the whole thing gives the impression of a cystic tumor. I have seen cysticercus vesicles removed from the tongue and nose, echinococcus vesicles removed from the back and thigh. The diagnosis of cysts was made in all the cases except in one of the latter where abscess was diagnosed, and in fact, instead of the customary encapsulation, there was suppuration around the dead echinococcus vesicle. I have introduced this as a sort of appendix, because we have nowhere else an opportunity of considering the parasites. The millions of *trichinæ* occasionally scattered through the muscles cannot be treated surgically, even when, according to the brilliant investigations of *Zenker*, the diagnosis may be, and has been, made in many cases. Dropsies of the subcutaneous-mucous bursæ and of the tendinous sheaths as well as spina bifida may also be readily mistaken for cystic tumors, if we do not attend to the anatomical seat of these swellings. Cystomata may also be mistaken for other gelatinous soft sarcomata and carcinomata, and for very soft fatty tumors. As stated, when an intention of operating renders a certain diagnosis necessary, we make an exploratory puncture. But what guides us chiefly, in the diagnosis, is the experience about the relative frequence of different tumors on different parts of the body; I have given you the sum of these experiences in each form of cyst, and in the clinic shall hereafter direct your special attention to this point.

As the above includes the *prognosis* of cystic tumors, all of which grow slowly when they exist as cysts without complication, we may pass at once to their *treatment*. We may remove cysts in two ways, viz.: by evacuating the contents, and locally applying remedies that may excite an inflammation which shall cause atrophy of the sac, or by extirpating the sac; the latter is always the simplest and most rapid, and we always give it the preference where it can be done easily and without danger to life. But in cysts of the ovary, thyroid, and other glands, that are deeply seated or from other causes dangerous, some other, safer operation is of course desirable, if it offers a prospect of success. We may induce shrinkage of the sac after precedent evacuation of the contents, by a suppurative or by a milder, drier inflammation. If you slit up the wall of the cyst its whole length, and keep the cut edges apart, there will be suppuration and granulation of the exposed inner wall of the cyst, with detachment of the portions of tumor or epithelium clinging to it; the sac then gradually shrinks up into a cicatrix, then decreases in size, and finally heals; but this may require months. You may attain the same thing in a more subcutaneous way, by ligatures or tubes through the tumor at different points; the irritation caused by these, as well as by the entrance of air, causes suppuration and granulation of the inner wall, and in favorable cases these may lead to atrophy; often this does not occur in the manner desired, or else it may require months or years; so that of these two methods the first is preferable; it is particularly applicable to cysts of the neck. We may attain shrinkage of the cyst and drying up of its contents in another way, namely, by puncture, with subsequent injection of tincture of iodine; we have already (page 579) said enough about the effect of this treatment. Here, too, the injection is followed by severe inflammation of the sac with sero-fibrinous exudation; then the serum is reabsorbed and the sac contracts. The latter method is particularly applicable when we have to deal not with contents of softened tissue, but with a fluid secreted by the walls of the sac, that is, chiefly with cysts whose contents are serous, and some sorts of mucous cysts. Cystomata developed from softened gelatinous substance and fat-cysts are not suited for iodine injections; for they are apt to be followed by severe inflammation and suppuration, with formation of gas, so that we are subsequently obliged to slit up the entire sac. And very thick walls, which contract very slowly or not at all, also contraindicate iodine injections. Hence among cysts of the neck we find some that are suited for this treatment, others which are not, because their walls are too thick. Of the ovarian cysts, too, unfortunately but few are suited for treatment by iodine injection, so that recently the extirpation of these tumors by laparotomy is considered the only certain operative proceeding; of late years the results from this operation have constantly been growing more favorable. Lastly, we must state that in some cases it is best to avoid any operation; for instance, I should consider it folly to persuade an old man, with a number of atheromata on his head, to have them removed; for, if the operation were followed by erysipelas, it might prove fatal.

## LECTURE L.

13. *Carcinomata.*—Historical Remarks.—General Description of the Anatomical Structure.—Metamorphoses.—Forms.—Topography.—1. Skin and Mucous Membranes with Pavement Epithelium.—2. Milk Glands.—3. Mucous Glands with Cylindrical Epithelium.—4. Lachrymal Glands, Salivary Glands, and Prostate Glands.—5. Thyroid Glands and Ovaries.—Treatment.—Brief Remarks about the Diagnosis.

### 13. CARCINOMATA—CANCEROUS TUMORS.

To give you an idea of how tumors were formerly diagnosed, and of the origin of many of the names still in use, I will read you a passage from the classical, and, in its time, most prominent, work of *Lorenz Heister*, the third edition of which, published in 1731, I have before me. Here (page 230) it says: "The name *scirrhus* is given to a painless tumor that occurs in all parts of the body, but especially in the glands, and is due to stagnation and drying of the blood in the hardened part." (Page 318) "When a scirrhus is not reabsorbed, cannot be arrested, or is not removed by time, it either spontaneously or from maltreatment becomes malignant, that is, painful and inflamed, and then we begin to call it *cancer* or *carcinoma;* at the same time the veins swell up and distend like the feet of a crab (but this does not happen in all cases), whence the disease gets its name; it is, in fact, one of the worst, most horrible, and most painful of diseases. While the skin remains intact over it, it is termed *hidden* (cancer occultus), but, when the skin has opened or ulcerated, it is called *open*, or *ulcerated cancer;* the latter usually succeeds the former."

It is not long since men lived in the simple belief that there was something real and truly practical in this mode of comparison and description. In a hundred years will they laugh at our present anatomical and clinical definitions, as we now do at good old *Heister?* Who knows? Time moves on with giant strides; things come to light, and, before we have time to look around, they are turned into history by the careful labors of energetic young experimenters.

In the natural sciences we always dislike to give short definitions, because this is often impossible, on account of the passage of one process, or of one formation, into another. We may say that carcinomata are very infectious tumors, and that this infection, which first attacks the lymphatic glands, afterward more distant organs, is probably due to the passage of elements from the tumor (whether of cells or juice is not yet known) through the lymphatic vessels and veins into the blood.

This common clinical definition of carcinoma should be controlled by the anatomical structure of these tumors. Anatomical peculiarities, easily recognized with the naked eye or with the microscope, are sought for. The classical monographs of *Astley Cooper* on diseases of the testis and breast (the latter, unfortunately, unfinished) show that, by a careful study of the points perceptible to the naked eye, a great deal may be attained by studying a single organ; but a generalization by aid of the anatomical preparations alone is impossible, as we have often felt, in the course of these lectures—it is frequently difficult, even with our present aids; so that I cannot blame *Virchow* for trying, in his great work on tumors, to give most minute descriptions of the different forms of tumors at certain localities. Here, where we must express ourselves briefly, to give our descriptions an anatomical basis, we must be somewhat more decided and summary. When the naked eye no longer sufficed for the diagnosis of tumors, the aid of the microscope was invoked, and characteristic appearances were sought that might occur in the same way in all the tumors we have described. Still, whether the characteristics of the cellular elements were sought in their processes, the size of the nucleus or of the nucleolus, the clinical and anatomical peculiarities would not always remain congruous. When the cells proved inefficacious as evidence of carcinoma, it was sought for in the general structure of the tumor; *alveolar formation* was asserted to be the anatomical peculiarity. We even come in collision with this idea occasionally; the net-like formation of neoplastic lymphatic gland-tissue may also be termed "alveolar," and even acknowledging that the lymphoma net-work is so peculiarly characterized by its form that it may be readily excluded, there still remain some forms of chondromata and sarcomata, especially the giant-celled, and other large-celled sarcomata forms, which we have already designated as alveolar sarcomata (pages 717 and 720), as the ghosts of cancer.

Since anatomical study, especially the origin of neoplasms, has been regarded as an essential principle of division, we escape all the difficulties just enumerated. Now, anatomy alone decides what is to be called cancer. In the clinic we then have to investigate how cancers of different formations and compositions usually conduct themselves: if they be infectious or not; whether they run their course slowly or rapidly; if they are usually solitary or multiple; where most frequent, and how they are most successfully treated. Most modern pathologists agree in calling only those tumors true carcinomata which have a formation similar to that of true epithelial glands (not the lymphatic glands), and whose cells are mostly actual derivatives from true epithelium. I am convinced that this view will constantly have more adherents, and that thus the differences about the anatomical definition of "carcinoma" will constantly diminish. Those investigators who, during the last few years, with all the modern aids, have worked without prejudice on this portion of the study of tumors, recognize the great importance of epithelial proliferation in those tumors that we call cancer, still most of them seek for a compromise between the different histogenetic views, and wish still to admit, in a modified form, the development of true glandular and epithelial cells from connective tissue (heterology proper) (*Rindfleisch*, *Volkmann*, *Klebs*, *Lücke*); only *Thiersch*, and recently *Waldeyer*, maintain, as I do, the strict boundary between epithelial and connective-tissue cells. *Waldeyer* defines carcinoma as an atypical epithelial neoplasm. But we must here state that in cancer-tumors, besides the epitheliums, there are usually numerous young, small round cells which, infiltrated in the connective-tissue portion of the tumor, form an important part of it. This small-celled connective-tissue infiltration, which exists in varying quantities wherever epithelial proliferations grow into the tissue, appears to be caused by a sort of reaction, and to be the result of the penetration of the epithelial new formations into the tissue, according to the number of infiltrated cells and their future fate, as well as the degree of vascularity, just as in inflammation it sometimes leads to softening, to atrophy, and cicatricial thickening of the tissue. In some cases this small-celled infiltration is so considerable as almost entirely to hide the epithelial new formation (from which it may be very difficult to distinguish, if the latter be small). We may then be in doubt if it should not be regarded as entirely independent, and occasionally, perhaps, as the sole constituent of cancerous tumors. Formerly I myself thought it necessary to agree to this, and even supposed that this component of carcinoma possessed a spontaneous power of infection; but further observations with new aids have made it appear to me more probable that, even in the smallest cancerous nodules, epithelial elements are proliferating. The epithelial cells, and the base on which they grow and from which they draw their nourishment, are most intimately connected. Many observations show certainly that the cellular infiltration of the connective-tissue base causes an increased proliferation of the superjacent epithelium; so it would not be difficult to suppose that the first impulse to the atypic adenoid proliferation was due to an irritative state of the epithelial base. But it is just as possible and probable that the epithelial proliferation is, as we usually consider it, the first formative process in the development of carcinoma. There can be no direct observation on this point; the connective-tissue infiltration is always there as soon as the epithelial proliferation; this so much impedes investigation of the first stage, that a choice of very favorable objects (such as flat cancer of the skin) alone will give any evidence in favor of our view, while the study of more difficult objects (as infiltrated lymphatic glands) in which, during life, the most varied cells are mixed up, will find plenty of support for *Virchow's* view (which I formerly held), according to which epithelial cells may result from proliferation of connective-tissue cells.

It is especially important, anatomically, to make a distinction between adenoma and carcinoma, as the two forms of tumors have some points in common. Pure adenomata are composed of newly-formed gland-substance which is entirely analogous to or at least very much like the normal; the connective tissue around the newly-formed acini has the same relation to them as to the normal.

In adeno-sarcoma there is little if any new formation of glandular acini, but the sarcoma merely encloses the glandular spaces which have remained normal, or are dilated. But it is characteristic of carcinoma that the epithelial covering of a skin or mucous membrane, or the epithelial lining of glandular cavities, grows into the skin, and even deeper, in the form of roundish nodules (acinous), or of round cylinders or rollers (tubular), just as occurs in the fœtus. While so doing, the epithelial cells usually preserve their form, only they often grow much larger than normal. The form of the glands from which these formations proceed generally remains typical for the neoplasm also; but it remains in irregular forms of glands, it is only rarely that cavities are formed, and that actual secretion goes on in these cavities. Besides the epithelial parts of these tumors, the connective tissue, bones, muscles, etc., into which the epithelium enters, conduct themselves as follows: We sometimes find them of normal, again of abnormal firmness, sometimes very soft, almost mucous, ordinarily in less quantity than the epithelial masses. It is usually pervaded by small, round (lymph) cells, often to such an extent that scarcely any fibrous tissue is left; generally the infiltrated small cellular elements are scattered diffusely in the cancerous (connective-tissue) framework; very rarely, we find numerous cells, collected together in a fissure between the connective-tissue bundles. When the tumor advances into the bone, the latter is eaten away, as in caries. I have not been able to satisfy myself that there is any new formation of connective-tissue filaments in the nodular and infiltrated forms of these tumors, nor have I been able to find any osseous new formation; but there is no doubt that such a new formation occurs in the papillary and villous forms, of which we shall hereafter speak. From this description you see that *Waldeyer's* expression about the epithelial formation in carcinoma being *atypical* (*tissu hétéroadénique* of *Robin*) is also well suited for distinguishing carcinomata from adenomata, as typical new formations.

As regards the vessels in these tumors, we may satisfy ourselves, by artificial injections, that the dilatation and new formation, by tortuosity and looping, are considerable; only the connective-tissue portions of the tumor are vascularized, the epithelial portions remain free; this is a very important anatomical criterion, as is the fact that true epithelial cancer-cells never unite together as the large epithelio-cells of some sarcomata do; *Waldeyer* has justly attached great importance to this latter point. I cannot go any further into the general histological description of these tumors, and hope that they may be recognizable from the above, although I acknowledge that it is sometimes very difficult to distinguish carcinoma from adeno-sarcoma and alveolar sarcoma.

According to my whole histogenetic view, I must regard it as impossible for an epithelial cancer to occur primarily in a bone or lymphatic gland. The observations that I know, to this effect (in the lower jaw, on the anterior surface of the tibia, in the lymphatic glands of the neck), do not seem to me sufficient proof, because the skin and mucous membrane are so near; there may have been an insignificant carcinomatous disease of the skin or mucous membrane as a starting-point of the disease, without its having been noticed.

The appearance of the cut surface of this tumor, and its consistence, vary so, that no general description can be given of it.

In the great majority of cases, carcinoma appears in the form of nodules; also as indurations of otherwise soft tissues, or as papillary proliferations. Rarely, the diseased parts are separated from the healthy tissue by a connective-tissue capsule; but, in most cases, the passage from healthy to diseased tissue is more gradual. In some cases there is no cancerous tumor, but a cancerous infiltration, there being no enlargement, possibly even a diminution in size of the affected organ. It is also characteristic of carcinoma that part of the new formation is very short-lived, disintegrates directly or after precedent fatty degeneration is reabsorbed, and then the infiltrated fibrous tissue contracts to a firm cicatrix. Besides this cicatricial shrinking, and not unfrequently along with it, there is often softening: it is, perhaps, even more frequent than contraction; at all events, it is more extensive. This softening is mostly preceded by fatty degeneration of the cells and caseous metamorphosis; central softening, opening outwardly, formation of a putrid ulcer, with fungous edges, is very characteristic of carcinoma. Mucous metamorphosis of the cell-protoplasm also takes place in some glandular carcinomata, relatively most often in those of the liver, stomach, and rectum; in rare cases, this also affects the connective-tissue stroma. This mucous cancer is also called *gelatinous* or *colloid*. When cancerous degenerations occur on the surface, the papillary layer may develop so as to become very prominent, as in some *papillary cancers* (destructive papillomata) of the mucous membrane of the lips, stomach, and portio vaginalis, and as in *villous cancer*, which develops on the mucous membrane of the bladder, in the form of dendritic, branched, large papillæ. If the cicatricial contraction predominate in a carcinoma (as it does in some forms of cancer of the breast), hard tumors or ulcers are developed, which have for ages been called *scirrhus*. Some carcinomata are brown or black, but still *melano-carcinomata* are rare. Most soft melanomata are sarcomata.

[Before true infection occurs in the lymphatic glands they are temporarily swollen by inflammation. This is shown, among other things, by the fact that after removal of some carcinomata there is complete and permanent recovery, although at the time of the operation enlarged lymphatic glands in the neighborhood were not removed. The glands form a barrier to protect the organism against infection; but in some cases the barrier is broken through, and metastatic tumors occur in internal organs and in the bones. In the lymphatic glands the infectious elements of carcinoma find most favorable soil for exciting epithelial proliferation of the tissue-cells.

In most cases no causes are known for the development of carcinoma; but in many cases there was precedent local irritation, that is, the neoplasia developed on an already pathologically changed base. Recent statistics notice local irritations at the point of development of about twenty per cent. of cases. Not to go further into details, I may mention examples of development of carcinoma from surfaces that have suppurated for years, such as fontanels, fistulæ, etc., from warts, lupous infiltrations, etc. Inheritance seems to be a cause in some cases, but there is not enough evidence on this point. Tendency to disease of the same system, especially, seems inherited, without the same organ being always attacked by carcinoma. This is oftenest seen in carcinoma of the sexual organs in women, e. g., cancer of the uterus in the mother, of the breast in the daughter, and the reverse. In females carcinoma is most frequent in the glands, in men in the skin.

I cannot confirm the common statements about cancerous cachexia: these patients finally become marasmic, like others having severe disturbance of important organs, indigestion and hæmorrhages; they emaciate rapidly and grow yellow or brownish. The general condition is most affected in cancer of the tongue, stomach, etc.; but where it does not bleed, decompose, or interfere with function, the patient does not become cachectic. Some pathologists even consider cancer a sign of superabundant health; without adopting this idea, we may say carcinoma is a local disease, not a symptom of a general affection, as a gumma is of syphilis. Formerly, cancerous diathesis was so firmly believed in, that the cure by operation was discredited; but to-day the belief has changed. Experience has not confirmed a popular belief that cancer is infectious.]

The view that epithelial cells may result from proliferation of connective-tissue cells also finds supporters among the new school, who either do not recognize the typical formation of the tissue from the germ-layers, or else do not acknowledge its significance for the pathological neoplasiæ. Since this question was first earnestly discussed it has come up repeatedly, not only in the same generations, but to the same person. I cannot here repeat all that I have said about the origin and increase of true epithelia; I will merely add that the carcinomatous and epithelial forms found in primary cancer are also invariably found in the infecting tumors in the lymphatic glands. This seems to speak strongly in favor of the traveling of cellular elements, for it is scarcely probable that the fluid from a columnar epithelial cancer should influence the cells in the lymphatic glands to produce cylindrical epithelium.

From the peculiarities described (page 684), even in the most difficult cases, we may always find the genetic differences between sarcoma and carcinoma. The first commencements of sarcoma and carcinoma are often scarcely distinguishable (compare Fig. 139 B with 162 and 163); both cases are very glandular in formation. But things change after a time; the cell-cylinders of the sarcoma have either started from vessels, or vessels soon grow in them; while this never happens in carcinoma, but the cylinders, even when quite large, remain without vessels, or else a cavity forms in them as in the development of glands (compare Fig. 139 B with 169).

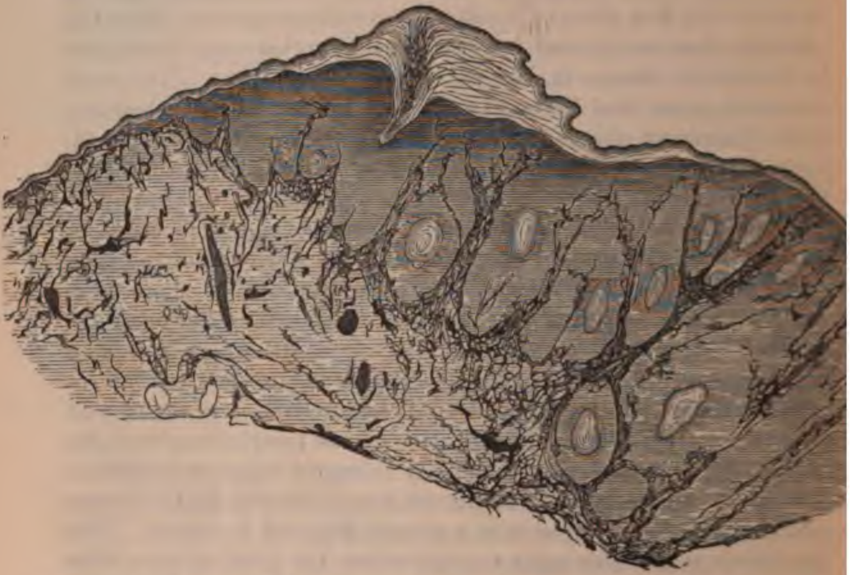
I dare not enter further into the general histological description of these tumors, and hope you will be able to recognize them.

When speaking of sarcoma I said something about the difference of its course from that of carcinoma. I here repeat again that the latter always first affects the adjacent lymphatic glands; often the infection does not extend beyond them; in other cases there may be metastatic tumors in internal organs or the bones. The small epithelial germs find the most favorable soil for their development in the lymphatic glands. The rapidity of the course varies exceedingly; this we shall consider more closely when treating of the topography of carcinoma.

[In some well-observed cases, patients who had been cured of cancer of the skin of the face, without having disease of the lymphatic glands, had carcinoma of such internal organs as are occasionally attacked primarily: thus, a man from whom cancer of the auricle of the ear was removed, had a cancer of the stomach running a rapid course; in a patient dying after a rhinoplastic operation for epithelioma of the nose, carcinoma of the intestines was found. In such cases, from absence of infection of the lymphatic glands, we cannot assume metastasis, particularly to organs where metastasis is very rare. Hence we must admit the possibility of a double disease affecting two distinct places in a patient disposed to cancer. This hypothesis might also apply to cases where ten years or more after the removal of a cancer another develops at a different point. Sixteen years after removal of cancer of the tongue there was cancer of the mucous membrane of the cheek, without recurrence in the tongue.]

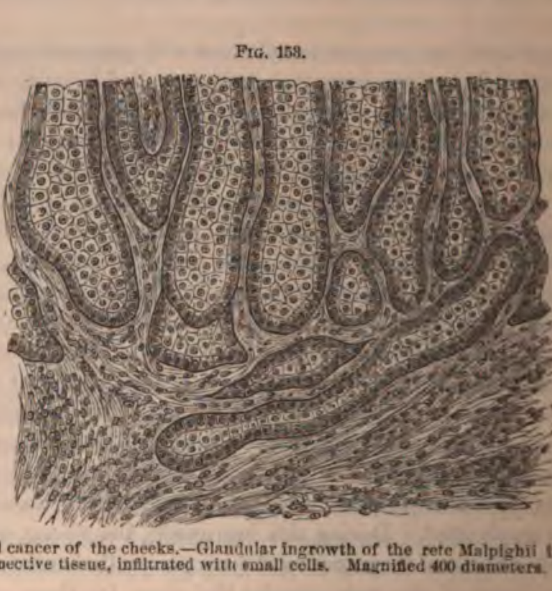
1. Skin (cutis) and mucous membranes with pavement-epithelium, Common *epithelial carcinoma* (specially so called because it was the first, and, until lately, the only form in which the main body of the cancerous tumor was known to consist of epithelium), or *cancroid* (cancer-like tumors; this name was chosen because these cancers of the skin were considered less malignant than those forms observed in the breast, which were considered as the type of true cancer). The cutis is covered by a layer of epithelium, from which in the fœtus there are various ingrowths into the subjacent tissue, namely, the hair-follicles, hair, sebaceous, and sweat glands. Mucous glands are formed on mucous membranes in the same way. Many assert that all these tissues may have epithelial outgrowths. I shall not deny this, but epithelial ingrowths may be most readily proved in the rete Malpighii. Next to this, a considerable collection of epithelium in the sebaceous glands and glands of the oral mucous membrane, and their enlargement, are also frequently witnessed; less frequently, the hair-follicles and sweat-glands are implicated. During this ingrowing, the young cells of the rete at first preserve their size and form; even their relation to the connective tissue of the cutis remains the same, for those cells lying

FIG. 152.



Commencing epithelial cancer of the vermilion border of the lip.—Growth of the rete Malpighii into the tissue of the lip.—Horny epidermis.—The blood-vessels injected. Magnified 60 diameters.

FIG. 153.

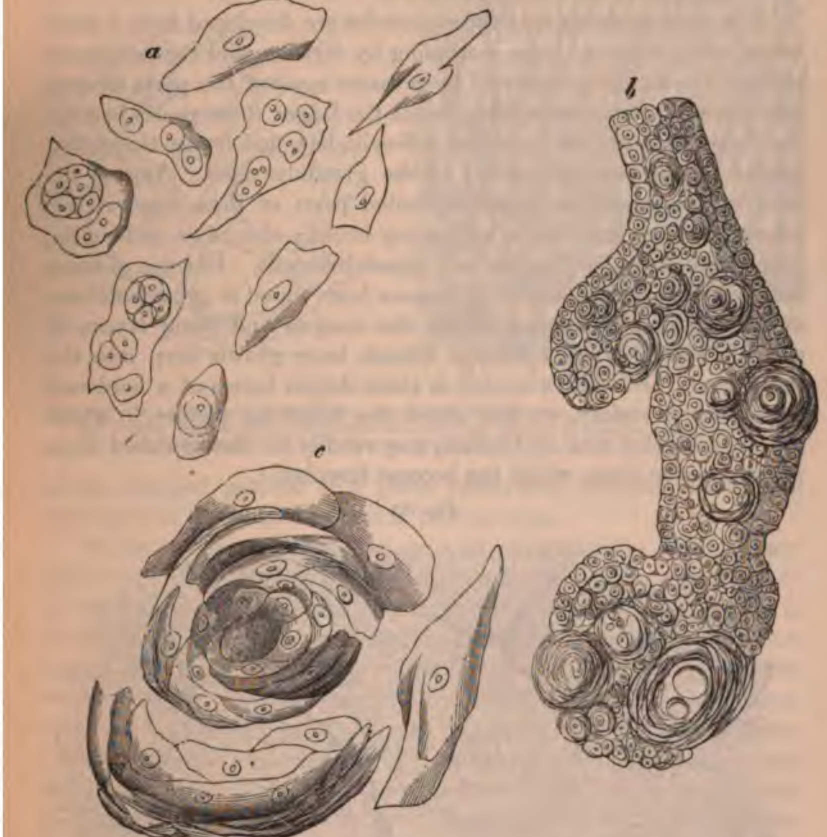


Flat epithelial cancer of the cheeks.—Glandular ingrowth of the rete Malpighii into the connective tissue, infiltrated with small cells. Magnified 400 diameters.

next to the connective tissue preserve a cylindrical form, just as on the normal papillæ of the cutis.

It is very probable that the epithelial, gland-like ingrowths not unfrequently grow into the spaces between the connective-tissue bundles where lymph circulates, for there the tissue offers least resistance. *Köster* thinks he has proved that all these tubes and cylinders lie solely in the lymphatic vessels. Although all his evidence in favor of this view is not tenable, it is still very enticing, for we might then readily understand why the adjacent lymphatic glands were occasionally infected early.

FIG. 154.

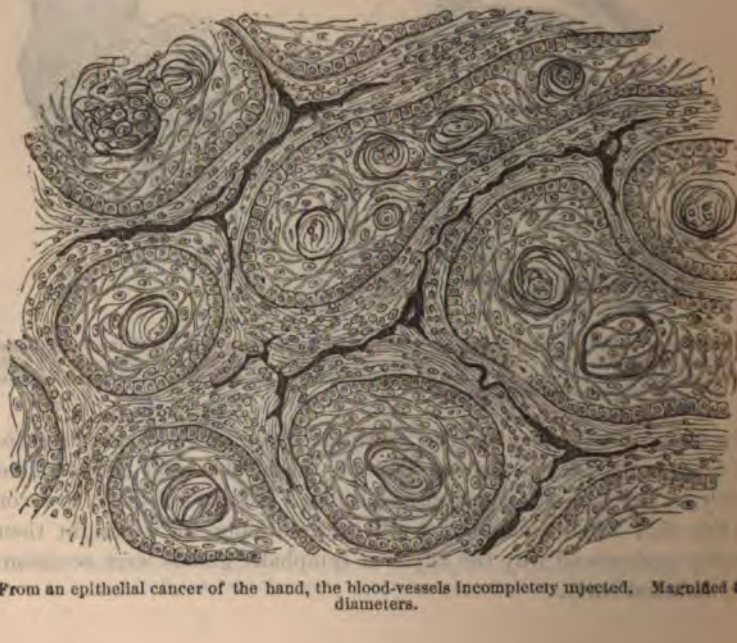


Elements of an epithelial carcinoma of the lip.—(Fresh preparation, with addition of very dilute acetic acid.) *a*, single cells with endogenous division of nuclei; *b*, a cancroid rod with concentric globules and outer cylindrical epithelium; *c*, an epithelial pearl that has been crushed. Magnified 400 diameters.

Subsequently, changes take place in these epithelial tubes; groups of cells unite and form globules, which gradually grow by the deposit of new cells of the form of flat epithelium, and thus form the cabbage-like, compound epidermis-globules (globules épidermiques, cancroid globules, epithelial pearls), which so much excited the astonishment of the first person that examined them.

It is most probable that these globules are developed from a number of conglomerated cells, increasing by division, and the peripheral layers of cells being flattened by pressure against the parts around, which are not very distensible; hence the larger these pearls become the more they project from the cell-cylinders, and hence they often appear at the terminal points of the glandular acini. Among the cells in the pearls, as in the epithelial parts of these tumors elsewhere, we often meet cells with many nuclei; also large cell-bodies, which have enclosed daughter and grandchild-cells. In some of these carcinomata stachel and riff cells have been found in great numbers, as in the boundary layers between the mucous and horny layers of the epidermis. If the epithelial masses have grown deep into the tissue, and if we make a section in these deeper layers of a hardened tumor of this variety, we find about the following picture, in which the alveoli, filled with epithelium, may readily be distinguished from the connective tissue which has become faveolate:

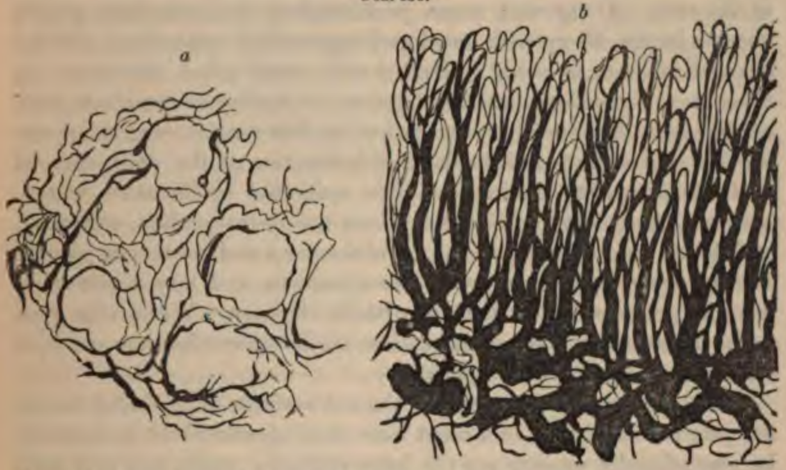
FIG. 155.



From an epithelial cancer of the hand, the blood-vessels incompletely injected. Magnified 48 diameters.

The vessels in this connective-tissue stroma assume about the shape in Fig. 156, *a*, while Fig. 156, *b*, shows a proliferation of vessels in the enlarged papillæ of a glans penis, as it occurred just at the development of the first epithelial proliferations.

FIG. 156.



Vessels from a carcinoma of the penis. Magnified 60 diameters. *a*, from the developed tumor tissue, vascular net-work around the epidermis pearls; *b*, vascular loops from the surface of the indurated but not yet ulcerated glans penis.

While in the last-mentioned case, as often happens, the papillary hypertrophy appeared at the very commencement of the development of the tumor as an essentially characteristic part, in other cases it is of an entirely secondary nature, i. e., the epithelial rods on the surface of the skin or mucous membrane soften, fall out, and leave the vascular connective-tissue portion in the form of a pouched ulcer, from which different papillary tufts protrude or subsequently grow. Carcinoma of the skin may begin as indurated papilloma, or as a wart, but just as often it begins as a nodule when the proliferation is at first circumscribed, grows into the skin; it enlarges slowly, without growing by apposition of new, small carcinoma nodules. The carcinomatous proliferation may also enter and grow through the cutis from a gradually-increasing surface, without causing any great prominence.

There is a decided difference between cancers of the skin, according as the epithelial proliferation enters the cutis more or less deeply; some cases remain quite superficial, scarcely entering the subcutaneous cellular tissue, and growing very slowly (flat epithelial cancer, *Thiersch*); others grow rapidly and enter the tissue deeply, destroying it (infiltrated epithelial cancer, *Thiersch*). The above description of cancer of the skin is from the infiltrated form; in flat epithelial cancer the outgrowing cell-cylinders rarely grow deeper than the deep layers of the cutis, and consist chiefly of the small, round cells of the rete. Along with these proliferations the sebaceous glands become larger, fill up with developed large-celled epithelium, and the connective tissue is richly infiltrated with small-celled elements. In these new formations the development of epidermis pearls is relatively rare. As viewed on the patient in this commencing stage, the whole forms a hard, slightly-elevated infiltration of the cutis, covered with desquamating epidermis. This epithelial proliferation is not, however, very solid; occasionally there are disintegration, softening, and detachment of the glandular proliferations and sebaceous glands. The highly-vascular connective tissue remains, and may continue to grow as granulations, or it may partially cicatrize. While this goes on in the centre of the new formation, the latter continues to grow, it may be very slowly, in the periphery.

At their very commencement, the cut surfaces of epithelial cancer are pale red and hard; in a short time they appear white and granular; occasionally we may see the large epithelial pearls and rods with the naked eye. Ulceration takes place from without inward, even more frequently than by medullary softening from within outward, and usually quickly follows their development. Mucous softening is rare in these forms.

In regard to the *topography*, we may mention the following regions of the body as the most frequent seats: (*a.*) *Head and neck;* here these tumors develop chiefly on the eyelids, conjunctiva, skin of the nose and face, the lower lip, oral mucous membrane, gums, cheeks, tongue, larynx, œsophagus, ear, and scalp. The first appearance varies greatly: the worst cases begin as nodules in the substance of the mucous membrane or skin, and quickly ulcerate from central softening; other cases begin on the surface; a fissure, crack, indurated excoriation, epidermoid scab, or a soft wart, forms; this at first apparently insignificant affection may remain superficial for a long time, slowly extending laterally, less so in depth, and having indurated borders. If the carcinoma develop from a wart-like formation, it may permanently preserve the papillary character. The parts once diseased are forever destroyed by the metamorphosis into cancerous tissue; in typical epithelial carcinomata there is no cicatricial shrinking; the ulcers which rapidly develop from these new formations vary, like other cancerous ulcers; sometimes smaller or larger shreds of tissue from the depths of the ulcer become gangrenous, leaving a crater-like loss of substance; sometimes the new formation proliferates, forming an ulcer with fungous, overgrowing edges. Not unfrequently, a cheesy pulp may be squeezed from this ulcerated surface; it comes out in a worm-like shape, just as the inspissated sebaceous matter does from the glands of the skin (comedones or maggot); this pulp is a mixture of softened epithelial masses and fat. Sooner or later, there is a gradually-increasing swelling of the neighboring lymphatic glands of the neck, which is not unfrequently painful; by degrees the glandular tumors unite together, or with the primary tumor; new points break out, and the local destruction gradually progresses; the new formation also extends in depth, destroying the bones of the face or skull, and taking their place. Death may result from suffocation or hunger, due to pressure of the tumor on the air-passages or œsophagus, or from pressure on the brain after perforation of the skull; more frequently, after gradually-increasing marasmus, it results from complete exhaustion, with the signs of excessive cachexia. On autopsy, we hardly ever find metastatic tumors in internal organs. All of these carcinomata on the head, face, and neck, are much more frequent in men than in women. The average duration of life of patients with cancer of the tongue and oral mucous membrane is a year to a year and a half. Cancers of the lips are radically curable by early and complete extirpation.

In previous works, I have termed the above form of flat carcinoma of the skin, "cicatrizing, atrophying, epithelial cancer, or scirrhous cutis," to define it more accurately from ordinary epithelial cancer. But now it seems to me better to make no special subdivision of it, hence I at once state that this is the mildest form of cancer of the skin, and, with few exceptions, attacks old persons; the disease occasionally begins as an infiltration of the papillary layer, with small nodules, always superficial; usually there is at first a local collection of yellowish epidermis, a small scab, after whose removal the skin appears at first only slightly reddened, scarcely infiltrated; when detached, the crust forms again; after repeated detachments, we find under it a small, rough, fine papillary, dry, ulcerated surface, which occasionally has, even at this period, hard, slightly-elevated edges; the small ulcer, on which new, dry crusts constantly form, extends through the cutis, but rarely into the subcutaneous tissue; its tendency is rather to spread laterally, occasionally it even heals in the centre, forming a cicatrix and new healthy epidermis, while a moderate induration and ulceration slowly progress in the periphery. In some cases there is no ulceration, only infiltration of the skin, with epidermis-scales and subsequent cicatricial shrinking.

The most frequent seat of flat epithelial cancer is the face, especially the cheeks, brow, nose, and eyelids; still other parts of the skin, which are subject to any form of epithelial carcinoma, may be attacked by this form; it is most frequent between the fiftieth and sixtieth year, and I find it as often in women as in men. Often the whole cutaneous surface, and especially that of the face and hands, appears very dry, and is covered by numerous dry, flat, yellow epidermis-crusts, as well as by numbers of small infiltrations, which often disappear again. This cancerous infiltration extends very slowly; occasionally it is six or eight years before a portion of skin as large as a dollar, or a side of the nose, or an eyelid, or portion of the ear, is destroyed; it rarely proceeds more rapidly. As the patients are generally old, they occasionally die of other diseases, and, for the same reason, there is often no recurrence after operation. But, even in cases not operated on or treated in any way, this form of carcinoma appears infectious in but few cases; the infection never extends beyond infiltration of the lymphatic glands, which does not occur till late, and then goes on just as slowly as the primary infection. Some writers have wished to banish this form of cutaneous cancer from the lists of carcinomata, and to place it among chronic inflammations as ulcus rodens (*Hutchinson*), or as a form of lupus peculiar to old persons. The various combinations of this neoplasia with distinctly-marked cancer in some points of the infiltrated edges, the possibility of its changing to proliferating cancer of the skin, and some other anatomical and clinical peculiarities, render it certain, in my opinion, that this form of infiltration and ulceration belongs among the cancers, and is the mildest and most feebly infectious among them.

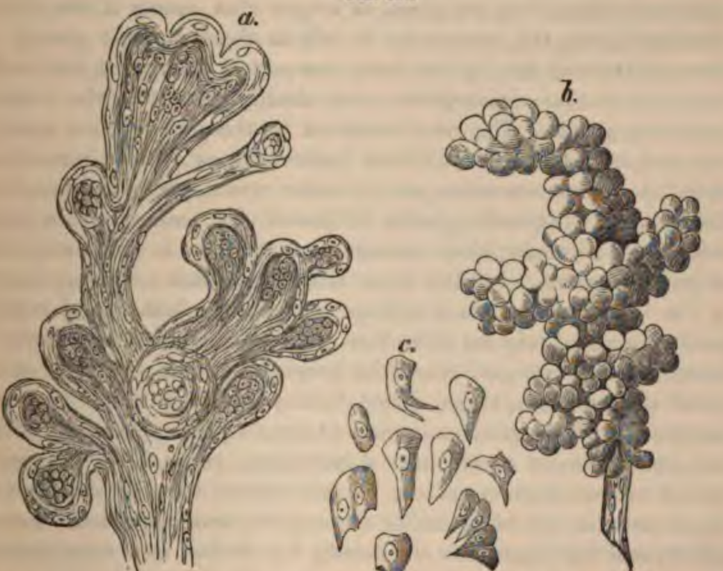
(*b*.) The second part of the body where this form of carcinoma is frequent is about the genitals. The portio vaginalis uteri, vagina, labia minora, and the clitoris, the penis, especially the glans and prepuce, are the parts most frequently affected. Of all these parts, the portio vaginalis uteri is especially liable to the disease, and here carcinoma ulcerates rapidly, and, as the surface of the tumor becomes deeply fissured and assumes the appearance of a cauliflower, this is often called cauliflower cancer, but, as sarcomatous papillomata may produce the same forms, this designation is uncertain. On all of the above localities the ulcerated tumor may have a destructive ulcerating or a fungous character, it may also be either infiltrated or superficial. The separation of uterine cancer is accompanied by very badly-smelling sanies, and often with repeated parenchymatous hæmorrhages. As regards the subsequent course of the disease, the retroperitoneal lymphatic glands are affected sooner or later; death usually results from marasmus; in these cases, also, we very rarely find metastasis in the internal organs, except in the neighboring glands which are directly infected.

(*c*.) Of other parts of the body that require the attention of the surgeon, we have to mention the hand, and especially the back of the hand. Not long since, I saw an epithelial carcinoma on the right forearm, which had developed from a fontanel, kept up for ten years with peas. I also saw an ulcer of the foot, which, after lasting for years, without any known cause became cancerous.

(*d.*) We also mention here the carcinomata growing from the vesical mucous membrane, which also has a pavement epithelium. Inaccessible as it is for surgical treatment, the surgeon must still be well acquainted with it, to enable him to make a differential diagnosis. It has already been frequently mentioned that papillary proliferations occur in carcinoma; this is particularly often the case in cancers on the inner surface of the bladder, which frequently grow in the shape of branched villi, and have consequently received the special name of "villous cancer."

Cancers starting from the cutaneous epithelium and glands have the same relation to villous cancer that adenoma has to papilloma. When papilloma assumes a peculiarly luxuriant growth, and at the same time epithelial masses grow into the part of skin affected, softening the connective tissue or muscle, in short, when the tumor assumes a distinctly destructive character, it may be regarded as carcinomatous papilloma or villous cancer. The boundaries between

FIG. 157.



Papillary formation of a villous cancer of the bladder, after *Lambl.* *a*, without, *b*, with epithelium; *c*, isolated epithelial cells of the villi. Magnified 350 diameters.

simple papilloma and villous cancer may be just as difficult to define as those between adenoma and carcinoma.

As above stated, a tumor like a mushroom forms on the inner surface of the bladder, growing into its cavity, and floating in the urine, its base being attached to the wall of the bladder, like a carcinoma, and its long, branched villi being covered with very large epithelial cells, while the ground-work of the papillæ is composed of connective tissue, whose meshes contain epithelial cell-cylinders, such as occur in carcinoma (Fig. 157).

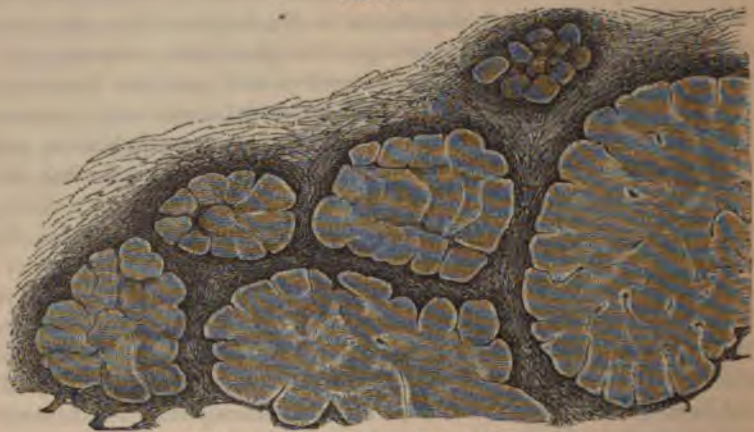
Now, a few words about the course of the above carcinomata as a class. They usually appear in elderly persons, say from the fortieth to sixtieth year, rarely later, but, unfortunately, it is not so rare for them to come earlier; I have seen cancer of the tongue in a boy of eighteen, and cancer of the uterus in a woman of twenty years. On the whole, country people are more subject to cancer of the lip than city people are. The earlier these carcinomata appear, the more proliferant the local tumor, the earlier the lymphatic glands are implicated, and the more rapid the whole course. It has often been observed that, after entire removal of the tumor, there is no recurrence. In some cases the disease runs its course very quickly, in a year; in others it lasts three, five, ten years, or longer (flat cancer of the skin); sometimes, also, the recurrence is only in the lymphatic glands, as when a cancer of the lip has been completely extirpated, but at the time of operation cancer-germs were already present in the cervical lymphatic glands. The new formation in the gland at first appears pale red, is a rather hard, diffuse infiltration, or a white kernel, but with time it becomes softer, and, to some extent, pulpy and purulent. The cervical lymphatic glands infiltrated with cancer have a great tendency to ulcerate; their microscopical structure is the same as that of primary cancer. I think there is no doubt that secondary cancer in the lymphatic glands is always due to transplantation of cancer-germs from the original focus (see page 671). The above forms of cancer scarcely ever go beyond the lymphatic glands; infection of internal organs (liver, lungs, spleen, kidneys) is very rare. The constancy with which carcinoma occurs at certain points, especially where mucous membrane passes into skin (vagina, penis, lips), has justly always excited much attention. It was natural to seek the causes of the disease in the structure of these parts, and in the irritations to which these openings were subjected; the dislike that most modern pathologists have to specific, unknown irritations has induced them to seek different reasons for explaining the obscurity about the specific causes of tumors of these parts. In regard to the lips in old persons, *Thiersch* attaches great importance to the fact that there, as in the cutis elsewhere, considerable changes take place with advancing age: there is decided atrophy of the connective and muscular tissues, so that the epidermis-formations, hair-follicles, sebaceous and perspiratory glands, as well as those of the lip, attain the preponderance, and receive most of the nourishment; hence all irritations affecting the lips (bad shaving, smoking tobacco, wind, bad weather, etc.) chiefly attack the glandular parts of the lip, and induce hyperplasia. In England, epithelial cancer often attacks the scrotum of chimney-sweeps (chimney-sweeper's cancer), from the irritation of the soot, it is supposed. These things may certainly have some effect, but it remains unexplained why they should be followed by cancers or infectious tumors, and not by chronic inflammations, catarrhs, etc. I shall not here follow this discussion further, but merely refer you to what was said about the etiology in the introduction to the section on tumors.

2. *Mammary glands.* I place cancer of the mamma here, as this gland is also a derivative of the epidermis, a cutaneous fat-gland on a large scale. The mammary cancers, however, differ greatly from those already described, and, although true epidermis-cancers occur in the breast, starting particularly from the areola, they are very rare.

Mammary cancer, which is unfortunately very frequent, seems to me almost always to begin with a coincident enlargement of the small, round, epithelial cells in the acini, and with small-celled infiltration of the connective tissue around them. With our present methods of examination it is impossible to tell whether the first changes occur in the gland-cells, or in the connective tissue; for the grouping of small, round cells about the acini soon becomes so excessive, that it constantly becomes more difficult to make out the further fate of the glandular acini. From my tolerably numerous observations on this subject, made by aid of the most improved methods, I think I may describe the following as the subsequent course:

The collection of cells in the acini leads first to their enlargement, which is occasionally accompanied by a trace of secretion (as is shown by the escape of serum from the nipple). As the collection of cells continues, there is more enlargement of the acini, and in such different ways, that we may distinguish an acinous (often large-celled) and a tubular (chiefly small-celled) form of mammary cancer. The former leads to the development of large, lobulated, glandular nodules; hence I call this the "acinous form," since in it the rough outlines of the acini are preserved. The following picture is a slightly-magnified one of the borders of such a tumor:

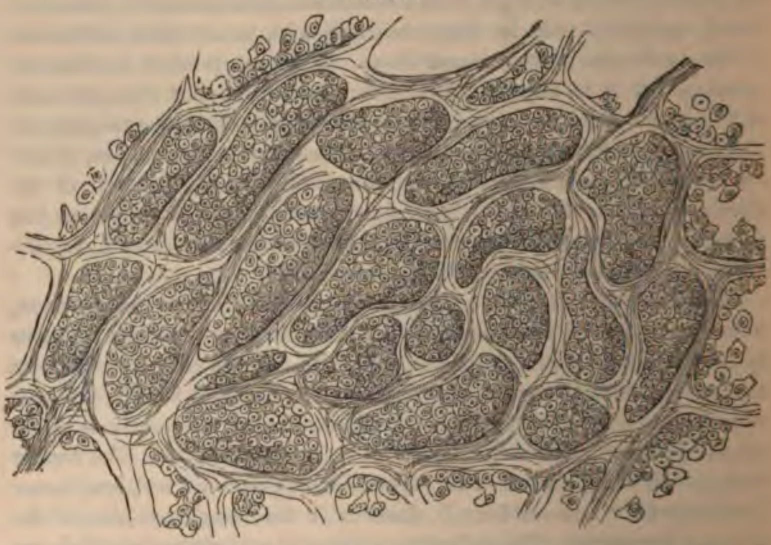
FIG. 158.



Mammary cancer, acinous form. Magnified 50 diameters.

The groups of epithelial cells, which are enlarged and grown to thick glandular clubs, are enclosed by infiltrated connective tissue, and traversed by a fine net-work of connective tissue (stroma), which I regard as the remains of the former partitions between the acini, but which others consider as mostly new formation.

FIG. 159.



Soft mammary cancer; alveolar tissue of the carcinoma; alcoholic preparation. Magnified 100 diameters.

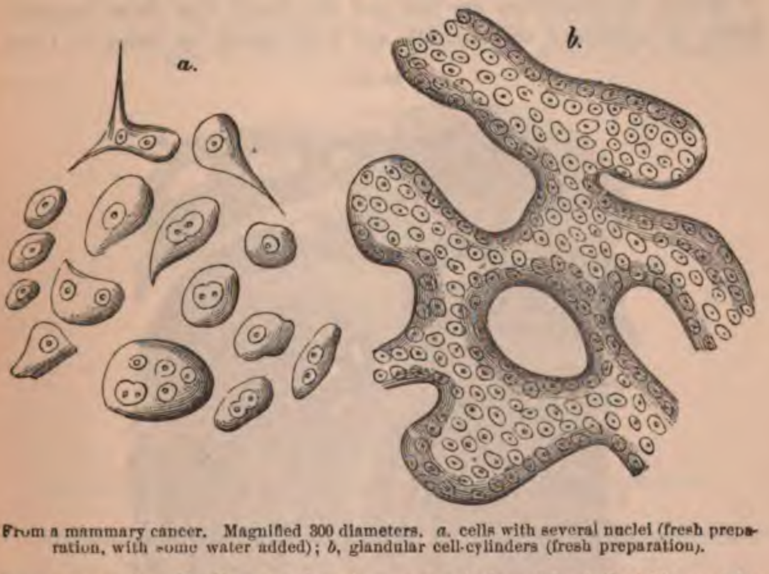
If we make a section through a hardened preparation of an acinous, soft, mammary cancer, when magnified more strongly, the tissue appears as above. I consider the cells in the large connective-tissue meshes as of epithelial origin (Fig. 159).

This variety of mammary cancer is mostly soft, granular on section, grayish white (medullary). If we scrape the cut surface of such a cancerous tumor, we readily evacuate a thick, whitish pulp; if we examine this while fresh, we find nodular cells, very pale, composed of large, many-formed cells with large nuclei; many of these cells contain several nuclei; they may perhaps be segregating.

The connective-tissue frame-work in which these elements were embedded, when empty, looks about as follows, if strongly magnified:

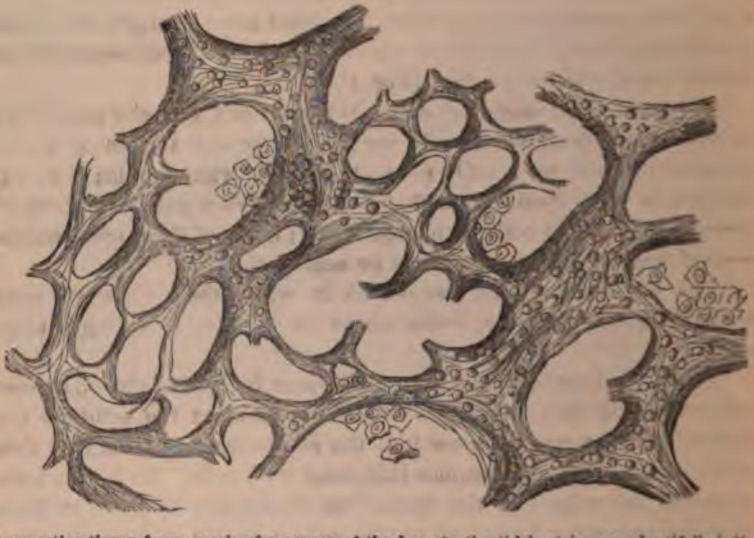
The second form, which is more frequent (is harder, and on section pale red), may be termed the "tubular" form, as the acini do not maintain their form, but grow into the connective tissue as very thin cell-cylinders, while it becomes infiltrated with cells. As in this form of cancer the cells from the epithelium do not usually grow so large as in the preceding form, and as the cells collected in the connective tissue occasionally lie together in groups, it is evident that it must be very difficult to decide which of these cancers come from the cell-masses of glandular epithelium, and which are pure derivatives of connective tissue, former wandering cells.

FIG. 160.



From a mammary cancer. Magnified 300 diameters. *a*, cells with several nuclei (fresh preparation, with some water added); *b*, glandular cell-cylinders (fresh preparation).

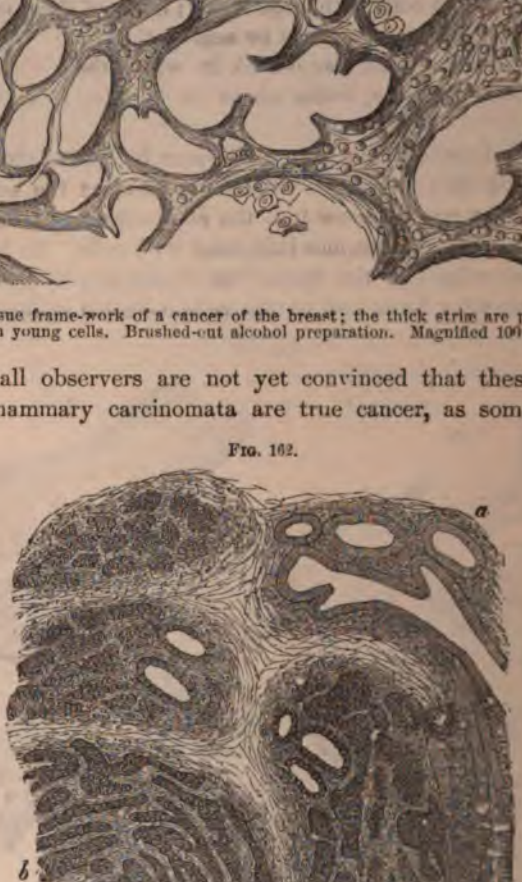
FIG. 161.



Connective-tissue frame-work of a cancer of the breast; the thick striæ are plentifully infiltrated with young cells. Brushed-out alcohol preparation. Magnified 100 diameters.

Hence all observers are not yet convinced that these frequent forms of mammary carcinomata are true cancer, as some of them

FIG. 162.

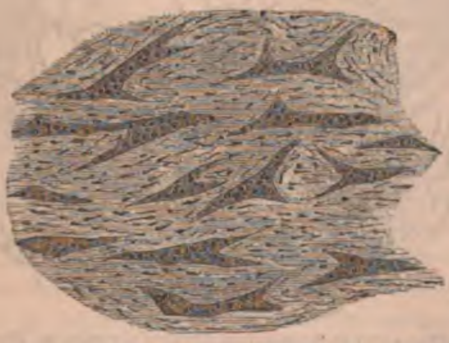


Cancer of the breast; tubular form. Magnified 150 diameters.

regard all the cells occurring here as derived from connective tissue. The final decision in this matter can only be made by the history of development; as long as we have no means of always distinguishing the young derivatives of epithelial cells from wandering white blood-cells, and the derivatives of connective tissue, we shall scarcely be able to say from every preparation whether this form of cancer of the mamma is more of an epithelial or connective-tissue nature.

Although all forms of cancer of the breast have a tendency to ulcerate, this is more the case in the softer than in the harder forms. The hardness of cancer of the mamma does not always depend on its richness in cells, but even acinous cancers that are rich in cells may be hard, if the cells are enclosed in tense connective-tissue capsules, as the normal acini are. The softening is central in nodules lying near the skin, or in the harder forms it is more frequently from without inward at points where the tumor presses against the skin and has become united to it. Mucous softening occurs rarely, mucous metamorphosis of the gland-cells is probably never seen. To the naked eye the softened spots appear whitish-yellow, granular (caseous, fatty softening) or grayish or dark red from vascularity, especially if there have been extravasations. By softening and encapsulation of the softened spot, which may be deeply seated, cysts may be formed in these carcinomata; retention and secretion cysts may also be developed in the mamma along with or in the cancerous tumor.

FIG. 163.



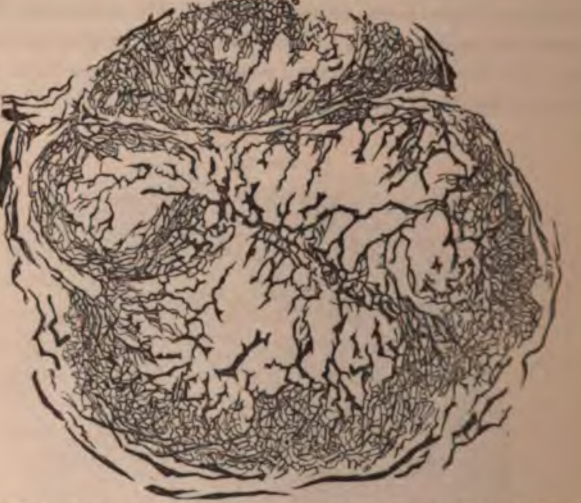
Cancer of the mamma, from a cicatricially-atrophied part. Magnified 200 diameters.

Atrophy is a very frequent process in cancer of the mamma; the nipple or other parts are thus retracted like the navel. On microscopic examination of these atrophied parts we see connective-tissue striæ with atrophied connective-tissue corpuscles, and the section of fine, branched canals (atrophied alveoli) which are filled with cell detritus or fat. This atrophy of the new formation is in some cancers of the mamma such an important factor, that it has given rise to a special form of cancer, "atrophying, cicatrizing cancer." It cannot be denied that in its pure form this variety of cancer has certain peculiarities which distinguish it from the ordinary, most frequent forms of cancer of the mamma; hence we prefer to describe it separately hereafter.

The development of cancer of the mamma is accompanied by considerable distention of vessels and new formation. In the youngest parts of the new formation there are numerous fine vessels and net-works of vessels; in the older, especially in the softening parts, the vessels grow wider, then are thrombosed and destroyed, so that, about points of softening in tumors, similar net-works of dilated vessels form as are developed on the formation of abscesses.

The following are the clinical symptoms of the development and course of ordinary cancer of the mamma. The disease usually begins between the thirtieth and sixtieth year, rarely earlier or later; the

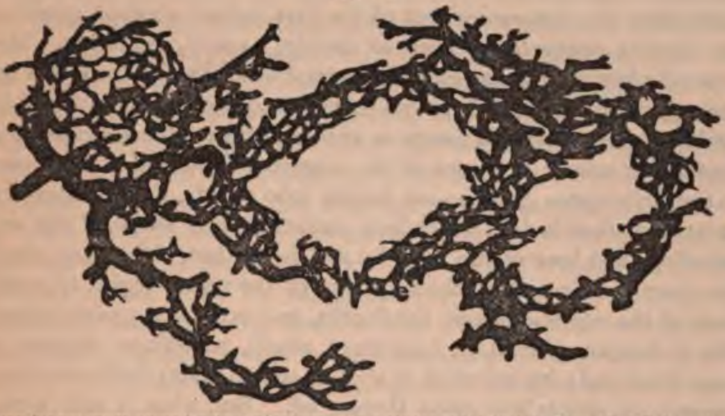
FIG. 164.



Vascular net-work from a very young cancerous nodule of the mamma. Magnified 50 diameters.

women attacked are usually otherwise perfectly healthy; married and unmarried women, fruitful and barren wives, of all conditions, are attacked. Not unfrequently the parents or grand-parents have died of carcinoma. Most frequently in one breast, especially in the outer and lower part, there forms a tumor, at first small and painless, that sometimes remains unnoticed for months; it is hard, firmly seated in the

FIG. 165.



Vascular net-work around points of softening in a cancer of the breast. Magnified 50 diameters.

gland, but at first movable under the skin and over the pectoral muscles; at first its growth is moderately rapid; possibly a year passes before the tumor reaches the size of a small apple; its volume is not always the same, occasionally it is larger and more sensitive, especially before and during the menses; but occasionally the tumor collapses somewhat, and is perfectly indolent. These symptoms are partly dependent on congestion of the mammary gland, partly on atrophy and cicatrization going on in the tumor itself. With time, in the course of some months, the tumor grows larger; the skin over it becomes immovable, and below it adheres to the pectoral muscle. The patients frequently do not notice the commencement of the swelling of the axillary glands, and, if the surgeon's attention be not occasionally directed to this region, the enlargement of these glands, which appears as a hard swelling of these parts, is not discovered till late; sometimes also these glands lie so deep and so high under the pectoral muscle that they are not felt till they have grown quite large. The lymphatic glands of the neck are less frequently affected in cancer of the breast; when they are, the prognosis is more unfavorable. If the progress of the tumor goes on undisturbed, the course, when moderately rapid, is as follows: The tumor of the mammary gland and those of the axillary glands gradually unite, so as to form a nodular, wavy, immovable swelling, which at some points adheres to the skin; the pressure of the tumor on the nerves and vessels in the axilla causes neuralgic pains and œdema in the arm; the patients, who previously had felt perfectly well, are compelled to keep in bed by the pain and swelling of the arm, which come on more especially at night, and have a piercing, boring character, while previously they may have been able to attend to their household duties. In this stage (say two years after the commencement of the first tumor) another symptom has usually appeared, or does so shortly, namely, ulceration. This generally begins with the following symptoms: Part of the tumor becomes prominent, the skin grows thinner and redder, is traversed by visible vessels; finally a fissure or vesicle forms on the elevated, red, fluctuating tumor; now part of the cancerous tissue which is exposed to the air becomes gangrenous, breaks into shreds, and a crater-like, excavated ulcer is left, which long maintains this shape, if the surroundings and base of the ulcer be still hard; but, if the parts about the ulcer be already soft, the substance of the tumor begins to proliferate at the edges and from the depths, and to cover the parts around like a fungus. An ulcer, sometimes torpid, sometimes fungous, is thus developed; its secretion is always sero-sanious; badly-smelling, gangrenous shreds are often thrown off. But, what is still worse, parenchymatous or even arterial hæmorrhages occasionally occur from the surface of the ulcer, and exhaust the patient. We have followed the condition of the patient till he has become partly or entirely bedridden; we now soon come to the catastrophe: the patient becomes pale and greatly emaciated; the appetite is lost, the strength grows less, the nights are often sleepless from the pain; opiates must be resorted to, to give the patients sleep and temporary relief. We now have the well-marked picture of *cancerous dyscrasia* or *cachexia*. It may go on in this way for months; the smell from the cancerous ulcer infests the chamber, the patients become weaker, the skin grows grayish-yellow and clayey. Pains on breathing and in the region of the liver, as well as in the bones of the limbs, come on. The patient becomes marasmic, and dies in agony after protracted, painful suffering, unless the end is hastened by pleurisy or peritonitis. On autopsy, in most cases we find carcinomatous tumors of the pleura, liver, and occasionally of the bones, it may be of the femur or of the vertebræ, or else of the ribs on the side where the tumor of the breast was. The whole disease has lasted two years and a half.

For many cases of cancer of the breast the above description will be very accurate, but there are some modifications of this course. First, the rapidity of the local course varies; the tumor may remain confined to the breast, without any affection of the lymphatic glands —a very rare case. The disease of the glands appears almost simultaneously with the tumor of the breast; this always leads us to expect a very rapid course of the disease, while conversely a very late and moderate local spread to the lymphatic glands indicates a mild, slow course of the whole disease. Carcinomata may come in the two breasts simultaneously, or in one soon after the other; this makes the prognosis much worse. In some cases there is no isolated tumor of the breast, but the whole gland, with the skin, becomes diseased at the same time. Lastly, an adenoma or an adeno-sarcoma may have existed eight or ten years, and then rapidly assume the character of a cancer, i. e., become immovable, painful, and accompanied by hardening of the lymphatic glands. Cases also occur where the tumor of the mamma diminishes so much that it is supposed it has entirely disappeared; unfortunately, this does not prevent the general outbreak of the disease, although it appears to retard it, or only to occur in mild cases, such as run on from four to six years. Some patients die early of anæmia from the ulceration and hæmorrhage, without any metastatic tumors having formed. The period for the occurrence of metastatic cancerous tumors in the internal organs also varies; generally, when the local growth of the tumor is slow, metastatic tumors appear late; still, there are exceptions to this rule. In cancer of the breast the localization of the secondary tumors is very regular, as already stated · the pleura, liver, and bones, are the most frequent seats of metastatic tumors.

The varying course of cancer of the breast renders it very difficult, indeed almost impossible, to compare the result of early or late operations with those cases that run their course without operation; even the age of the patient causes great differences: in old persons, the disease almost always runs a slower course than in young ones; numerous entirely unknown influences come in play. The most experienced surgeons have given very different opinions about operating, some declaring that the course of the disease is hastened by operation, others that it is retarded. The statistical tables that have been published aid little in solving this question, because cases of all sorts are thrown together in them; to obtain a correct result from them, the cases must first be separated on certain principles. But what good would this do? It would always be a question, in each case, whether we should aid the patient by an operation or not. The tumors will almost always return in the cicatrix, in its vicinity or in the neighboring lymphatic glands, because *they are usually operated on too late;* the patients will then die of metastatic tumors, if they are not carried off sooner by suppuration, hæmorrhage, or acute disease. How much does the patient suffer from the tumor? What danger does it induce locally? These are the first urgent questions. But I am anticipating by considering here the treatment, which we propose studying more attentively at the end of this section on cancerous diseases. Examination of the enlarged lymphatic glands, which partly adhere together, shows that the smaller are more succulent and vascular than normal;

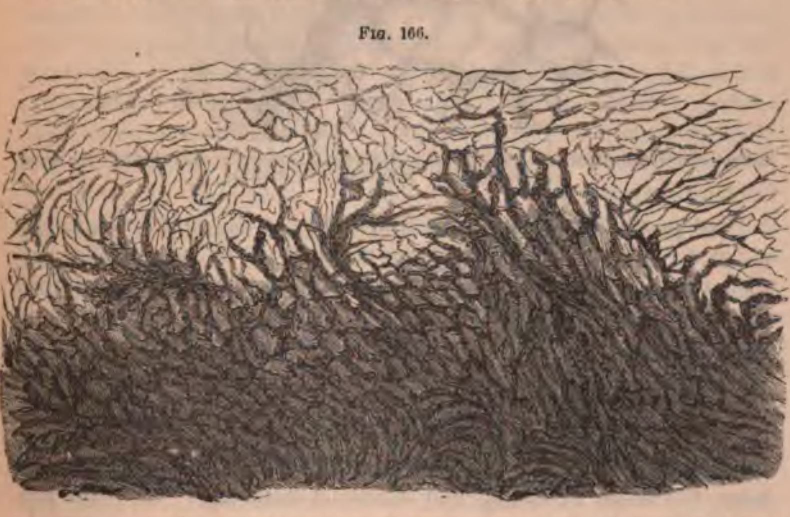
the larger contain hard white or grayish-white nodules, and are occasionally softened, caseous, and have a granular cut surface. On the whole, the lymphatic glands show the same characters as primary cancers; this also extends to the microscopic texture. Although it could probably only be proved in pigmented carcinoma that the first swelling of the lymphatic glands depends on transformation of tumor-cells into the lymphatic glands, still I consider the same thing true of all carcinomata; in some cases the epithelial nature of the new formation in the lymphatic glands is just as striking as in the primary tumor of the breast, in others such a distinction is impossible.

Carcinomatous nodules of the pleura, which develop after carcinoma of the breast from direct conduction of the seeds, are usually hard, pure white, and small-celled; the same is true of the external appearances of secondary cancer of the lungs and liver; but the latter are not unfrequently large-celled and acinous. Although I regard it as probable that these carcinomata are also due to direct emigration of carcinoma-cells or to transportation of the latter by the lymphatic or blood vessels, this cannot be proved.

Some cases deviate from the above course, as is shown by early and continued shrinking of the new formation. This form is called *scirrhus mammæ*, atrophying, cicatrizing, shrinking carcinoma, connective-tissue cancer. The picture of the disease and the anatomical changes will appear from what follows.

In the mammary gland, rarely before the fiftieth year, there forms a hard spot—we cannot say a swelling—but the hardening is rather accompanied by a partial or even a total decrease in size of the gland; this hardening usually forms without, rarely with severe pain; it comes on very slowly. If we now suppose the hardened glands removed and examine the diseased portion, we find the tissue so hard that we can scarcely cut it; to the naked eye, the cut surface shows a hard, fibrous cicatrix, with connective-tissue striæ gradually extending into the comparatively healthy parts around. In typical cases, except this cicatrix, we shall scarcely discover any thing pathological with the naked eye; but, at the periphery of some of these tumors we see a pale-reddish part with a fatty lustre, more marked in spots, lying between the cicatrix and the healthy tissue, and passing into both. If we examine fine sections of the cicatricial tissue after previously hardening it still more in alcohol, we find little besides connective tissue and elastic filaments; but the connective-tissue striæ have not the same peculiar regular course that they have in fibroma; they are irregularly intertwined, and, as above stated, they are accompanied by many elastic filaments, which rarely happens in fibroma. But examination of the bordering tissue gives the following: There is cell-infiltration, to a very slight extent, it is true; there is development of small groups of pale bodies, like lymph-cells, with single nuclei, as in the commencement of any new formation. Part of these cells are arranged in long groups (tubular), somewhat larger than the rest; these are doubtless derivatives from the epithelial remains of the shrunken glandular acini. All the cells of the neoplasm appear to be very short-lived, for they are scarcely formed before they commence to decay, without going on to further development; then the connective tissue, which has been somewhat distended, shrinks together, and, as a result of this process, we have the cicatrix; but peripherally this slight cell-infiltration constantly extends; hence complete, spontaneous disappearance of the new formation very rarely, if ever, occurs. If the borders of this tumor be inspected under a low power of the microscope, we see how the small-celled infiltration advances between the meshes of the connective tissue, and closely follows them.

FIG. 166.

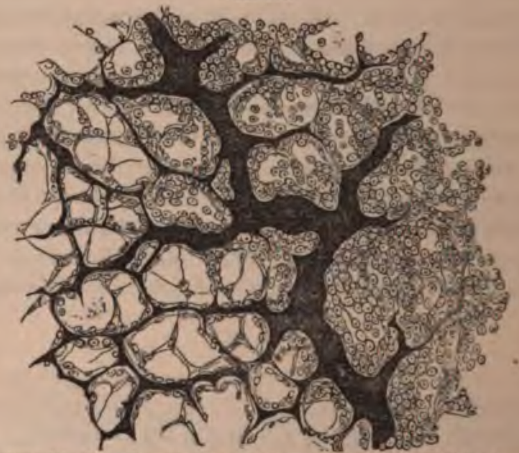


Connective-tissue infiltration advancing into the cutis from the borders of a cancerous nodule of the mamma; the dark shadings correspond to the advancing small-celled infiltration. Magnified 50 diameters.

The extension of this infiltration into the fatty tissue occurs just as in inflammation; most of the young cells are found in the vicinity of the vessels, so that we can scarcely avoid thinking that in these cases also white blood-cells escaping from the vessels cause the cellular infiltration.

As in these cases the infiltration of the connective tissue with lymphoid cells is very decidedly the predominant morbid process, while the epithelial proliferation is very secondary, I formerly tried to give this form of cancer of the breast the name of "connective-tissue cancer." But, as this has led to misinterpretation in regard to the modern anatomical understanding of carcinoma, I shall not try to preserve this term.

FIG. 167.



Cellular infiltration of the fatty tissue in the periphery of a hard cancer of the breast; the blood-vessels injected. Magnified 200 diameters.

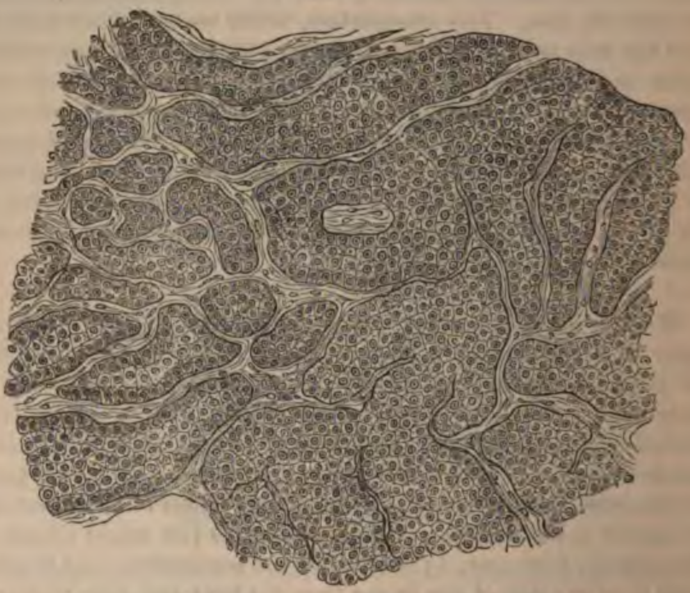
The peculiar anatomical and clinical course has caused some surgeons to strike this new formation from the list of tumors, and particularly from that of cancers. If we examine more closely the clinical course of these cases, we have already noticed that they usually only occur in old persons, and that the local disease progresses slowly; some cases last seven or eight years before half of one breast is atrophied. The general health meantime remains unimpaired. The lymphatic glands occasionally participate in the disease; in this case the process goes on just as in the mamma; there is very little enlargement, but much hardening and cicatricial shrinking. The more rapidly and completely the new formation atrophies, and the more slowly the process extends, the more injurious it is; after extirpation or cauterization this variety of cancer does not recur for a long time, if it does so at all; metastatic tumors are rare; in the main, the infiltration does not appear to differ much, anatomically, from that in chronic hepatitis and nephritis with subsequent shrinking; why, then, distinguish this scirrhus from those processes? *Wernher* terms this disease of the mamma *cirrhosis mammæ*. I recognize perfectly the justice of doubting the carcinomatous nature of some cases of scirrhous mammæ, but must still insist upon classing them generally among cancers, for the following reasons: As you already know, among tumors the process of contracting is peculiar to cancers; moreover, the contracting cancer is not unfrequently combined with ordinary cancer; indeed, it is more common for more or less cancerous proliferation to go on along with the scirrhous affection, while the wholly-cicatrizing cancers are relatively rare. This combination, which occurs neither in cirrhosis of the liver nor of the kidney, speaks entirely for the near relation of this cicatrizing new formation to cancer; in these combined cases there are also local recurrences of the extirpated tumors, tumors of the lymphatic glands, and even metastatic cancers of internal organs. In the tumors that consist *chiefly* of cicatricial substance, and hence are to be classed rather with scirrhus than with ordinary cancer, we may give a tolerable prognosis, inasmuch as the disease always runs a slow course.

We now mention another form of cancer of the breast which also begins as an induration in the gland, but soon extends to the skin, and there, in the form of small nodules, quickly spreads over the whole skin of the anterior wall of the thorax; the second breast is often affected the same way. This *cancer lenticularis* (*Schuh*), squirrhe pustuleux ou disseminé (*Velpeau*), appears partly as a primary, partly as a recurring form after extirpation of hard cancer of the breast, and not exactly in old women. This small nodular (we might almost say tuberculated) form may, by confluence and contraction, lead to actual lacing in of the skin of the thorax from the front and sides (cancer en cuirasse, *Velpeau*); the course is slow, the tendency to metastases to internal organs is not great, but the prognosis is very bad, because every attempt to prevent local extension by operation is in vain.

3. *Mucous membranes with cylindrical epithelium.* Most cancers that form in the nose and antrum Highmori, and gradually extend to the upper jaw, ethmoid and sphenoid bone, as well as into the orbit, start from the mucous membranes of the nose and antrum Highmori. The ciliated or non-ciliated epithelium of these membranes only extends to the openings of the mucous glands, and even in the development of cancers of the glands at these points rarely grows into the deeper parts. It appears to be rather the acini of the gland itself from which the proliferation proceeds, for these cancers appear to be mostly composed of acini or tubuli, which have small or larger round cells, rarely cylinder-cells, still more rarely ciliated cells. The shape of the newly-formed acini and their size here differ enormously, but often

are so distinct, so normal, that they may be mistaken for normal mucous glands; to render this deception complete, it not unfrequently happens that the newly-formed acini secrete mucus, which remains and collects in them. If the secretion from many acini be retained, the form of the neoplastic glandular acini be perfectly round, and the interstitial connective tissue be but slightly developed, the hardened, fine sections of such a tumor may very much resemble tissue of the thyroid gland. The interstitial tissue is usually very soft in these tumors; as in the corresponding mucous membranes themselves, it may be almost mucous. Interstitial papillary proliferations of hyaline vascular connective tissue (cylindroma) also occasionally occur here.

FIG. 168.

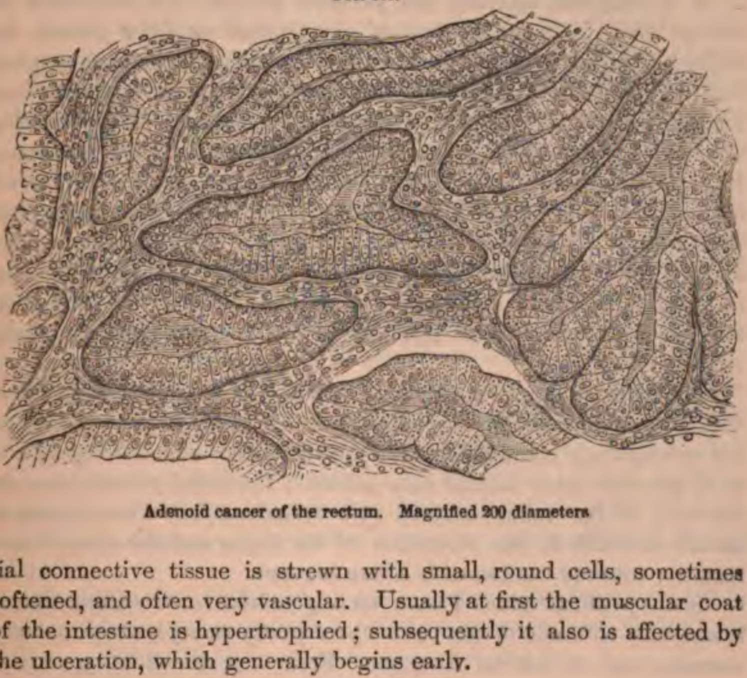


Cancer of the mucous glands from the interior of the nose. Magnified 200 diameters.

These tumors are always very soft, white, medullary, or gelatinous, except when very vascular; then they are dark red. The bones are destroyed by caries, without a trace of reactive bony new formation or osteophytes. The appearance and clinical course of these tumors are somewhat peculiar, differing from other carcinomata. They occur any time after the twentieth year, grow rapidly, and project sometimes through the nares, again through the cheeks or inner canthus of the eye; they are occasionally very sharply bounded or encapsulated, which may be known by palpation, and proved on operation; some times they are more diffusely spread in the upper jaw. In these mucous-gland cancers of the face I have never seen infection of the lymphatic glands, and am convinced that these patients could be saved by an early complete operation. In all the patients that I have operated on, I have never been satisfied that the tumor was entirely removed by the operation; it always projected too far posteriorly or upward to permit the operation to be completed with safety. Hence, I usually witnessed local recurrences, which proved fatal by marasmus or pressure on the brain, or else the patient died from the extent of the operation; in none of the cases examined *post mortem* did I find internal tumors.

In the stomach gland-cancers are frequent, especially with mucous softening (gelatinous cancer), and secondary cancer of the liver; cancer of the duodenum is very rare; of the parts of the intestinal canal attacked by this disease we are only interested in the cancers of the rectum. These are almost exclusively gland-cancers, and the proliferation proceeds from the large glands of the large intestine, which grow in the shape of tortuous and branched tubes; the calibre of the gland is often maintained, and they fill with mucus, and the cylinder-cells may maintain their form and become very large. The interstitial connective tissue is strewn with small, round cells, sometimes softened, and often very vascular. Usually at first the muscular coat of the intestine is hypertrophied; subsequently it also is affected by the ulceration, which generally begins early.

FIG. 169.



Adenoid cancer of the rectum. Magnified 200 diameters

As the first symptoms of cancer of the rectum are usually constipation, discharge of mucus, and slight hæmorrhage, these patients are mostly treated for some time as if suffering from hæmorrhoids, before the diagnosis is made by digital examination. Induration and nodular infiltration, leaf-like proliferations commencing close above the sphincter ani, soon extend to the whole circumference of the mucous membrane, so that a thick, prominent ring, a stricture of variable length, may be felt. This new formation can only be removed by extirpating the rectum. When the rectum is taken out, we generally find an ulcer with elevated edges and indurated base, and the parts around infiltrated with medullary substance; at some points also there are cicatricial contractions. The inguinal and retroperitoneal glands are affected rarely and late in the disease. The patients generally die from the stricture of the intestine, from marasmus, due to hæmorrhages, and putrefaction of the cancerous tissue.

Occasionally also cancers, composed mostly of cylindrical epithelium, start from the *pars cervicalis uteri.* These first attack the uterus, then the surrounding parts, and lastly infect and infiltrate the retroperitoneal glands; they combine with flat epithelial cancers, and do not differ from these in their course.

4. *Lachrymal, salivary, and prostatic glands.* The same kind of tumors grow from the *lachrymal* glands that we have already described as growing from the nasal mucous membrane, acinous glandular new formations, with soft, occasionally mucous, or even papillary hyaline interstitial connective tissue (cylindroma). They develop about the age of puberty, and are characterized by great tendency to local recurrence. All the cases of this nature that I have known of finally died from the local recurrence; it might be not for several years; neither the lymphatic glands nor internal organs were affected. *O. Becker* has described tumors of this sort, in which most of the glandular acini contained a certain quantity of mucous secretion, as also occurs more especially in the glandular cancer of the rectum.

The *salivary* glands may also be the seat of glandular cancer, but they do not come till old age; then, however, they grow rapidly, and not unfrequently resemble chronic inflammation. The newly-formed acini are often more tubular than acinous; epithelial pearls occur on the ends of the tubuli, covered with cylinder-cells. These patients usually succumb to the ulceration of the tumor and the general marasmus; internal carcinoma is a rare sequent.

In the *prostatic* gland I have seen glandular cancer a few times; it was very soft, and in one case where partly extirpated it was very vascular, and of acinous structure. From the excellent statistical work on malignant new formations in the prostate by *O. Wyss*, it appears that, in almost every case, these carcinomata also prove fatal solely from the local symptoms. Lymphatic glands and adjacent parts become infected; there are very rarely secondary cancers of internal organs.

5. *Thyroid gland and ovary.* I place these two organs together, as they both originate from true glandular epithelium, and both contain follicles, formed by choking off of glandular canaliculi. In cancerous disease both organs fall back into the embryonal type, i. e., the follicles grow again to tubes and canaliculi, from which again new follicles are developed; but some of these carcinomata, which are rare, consist entirely of cell-canaliculi, without any development of follicles. Young persons, as well as old ones, may be attacked by this form of cancer. Its course is usually rapid, for the cancers of the thyroid grow into the windpipe or close it by pressure, while the ovarian tumors are characterized by their enormous growth and rapid adhesions with the surrounding parts, and by the speedy development of ascites prove dangerous.

From variations in their course and anatomical structure we must separate the different forms of carcinoma; we may consider their treatment together. Treatment of the carcinomatous dyscrasia (carcinosis) is usually regarded as a *partie honteuse* of medicine. I cannot admit this. It is true we cannot cure the disease; but is not this also true of many other acute and chronic diseases? Can we arrest a cold in the head at any stage? Can we check the course of the acute exanthema or typhus? Can we cure tuberculosis? Certainly not; in all these cases, as in many others, the disease runs its typical course; we give little medicine, at least we avoid all heroic remedies. In carcinosis our therapeutic impotence only appears so great because the disease almost always proves fatal, and we can do nothing to oppose its course; in fact, our treatment is as inefficacious in coryza as in carcinosis; but the former is not a fatal disease, hence no special demand is made on the physician. We have become accustomed to failing to cure cold in the head; we must grow accustomed to the course of cancerous as to that of some other diseases; this will not interfere with our sympathy for these poor patients, nor must it prevent our striving for increased knowledge and improved treatment of the disease. I think that much may yet be attained in this direction.

The indications for treatment are to remove the cancerous tumor as soon as possible, so as to avoid infection, or at least obstruct its course, and thus diminish the evils accompanying it.

As long as cancer has been known, remedies for it have been sought; there is no active medicine, no form of dietetics, or mineral springs, that have not been recommended for cancer, and, to some extent, actually believed in. I should have to root up the entire old and new materia medica if I would tell you of every thing that has been thought and written on this subject. Like all incurable diseases, carcinosis also has been a wrestling-place for the charlatan, and even of late years Italians and Americans have claimed to cure the disease by special nostrums. Unfortunately, all these are deceptions, or at least what part of it is true has been long known.

Unfortunately, the *etiology* of cancer gives no clew to treatment; we know too little of the causes why certain tumors are so infectious, while others are not so. A blow, kick, etc., may occasionally induce an outbreak of the disease in some few cases, but cannot excite the predisposition to cancer. In some cases inheritance of the disease is evident. Care and anxiety may hasten the course of the disease, but do not induce it. All this is of no avail for the treatment. There is no specific for carcinosis; but by this we do not mean to say that all internal treatment is unnecessary or useless. By no means. The disease should be treated internally whenever there are indications for treatment, or any symptoms pointing to the use of certain remedies. As anæmia is not unfrequent in cancerous patients, iron in various preparations, or chalybeate mineral waters, may be employed. Occasionally, in persons with faulty nutrition, cod-liver oil, etc., as well as bitter medicines, prove beneficial by aiding digestion. Very debilitating treatment, by sweating, purging, mercurials, etc., is to be avoided, for life will be preserved the longer the more the strength is maintained. Among the mineral springs, the active ones, such as Aix-la-Chapelle, Wiesbaden, Karlsbad, Kreuznach, and Rheme, are injurious; only the milder indifferent thermal springs, such as Ems, Gastein, Wildbad; also, milk and whey cures, strengthening mountain air may be recommended without injury, if their use seems on other accounts desirable. Residence in southern climates is usually of little benefit for cancerous patients. Toward the end of life, when debility is increasing, a strengthening, easily-digested diet is important; and lastly, as the pain increases, the skilful use of various narcotics relieves the sufferings and death of the patient. The disease of internal organs may offer special indications to which I shall not here refer. Arsenic has been frequently tried, and with apparent benefit in a number of cases; it should be given till signs of its action are observed.

As regards *external* treatment, the first thing always is the removal of the tumor, if this is admissible, from its locality. The operation may be done with the knife or caustics; the ligature or écraseur can scarcely ever be employed here (the latter, perhaps, answers only in amputating the penis or tongue). But, before passing to the choice of either of these methods, we must consider the question, whether it is advisable to operate at all, even if it can be done easily and without danger to life, for the views of experienced surgeons differ on this point. Some surgeons never operate for cancer. They assert that the operation is always in vain, because the disease recurs; if the recurring tumors be operated on, new recurrence takes place the sooner; these surgeons even assert that, the more we operate locally, the sooner secondary lymphatic tumors and metastatic cancers form, the local tumor acting as a sort of derivative for the tumor-disease; that this product of disease cannot be removed without favoring the outbreak of the disease elsewhere; that, if we nevertheless wish to remove the tumor, we should lead the morbid juices to some other point, as by establishing an artificial ulcer by means of a fontanel or seton. Concerning this view, which comes from the old humoral pathology, we may say that it remains unproved, and is partly also disproved by experience. We consider it as demonstrable by daily experience that the glandular swellings are essentially due to the development of the primary tumors; we have already stated our belief that the participation of the lymphatic glands in carcinoma is, according to all analogy, caused by local contagion, let the process be what it may. When cases occur where, after removal of cancers of the breast or lip, swellings of the lymphatic glands appear, though previously imperceptible, we must consider that the commencement of the disease was so slight as to escape observation.—How far the existence of a primary and secondary cancer of the lymphatic glands influences the subsequent course of the disease, the appearance of metastatic tumors and general cachexia, is a question which cannot be answered, because the disease does not run its course in a regular time; if it did, we might form a rule as to the advisability of operating, by comparing cases that were operated on with those that were not. Approximate results might be attained by classing together cases that were alike in age, constitution, variety of the tumor, etc.; but, as the accurate distinction of the varieties of carcinomata, and consequently an exact arrangement of the cases, has only lately been attained, and even now is not generally known, we cannot at present expect much in this direction; individual observations rarely suffice for definite conclusions. The experience from carcinoma of the face, that the most extensive disease of the lymphatic glands is very rarely accompanied by metastatic tumors, strongly favors the belief that the disease is not made more active by these strongly-developed local tumors, and that carcinomata of the lymphatic glands do not increase the predisposition to metastatic tumors.—In reply to the question, whether carcinoma should ever be operated on, we may say that operation probably has no direct influence on the diathesis, and that the operation, if done at all, must be done for other reasons. We said intentionally that the operation has no *direct* influence on the course of the disease, but we think it has an *indirect* influence, as the tumor induces other causes of disease; the weakness, anæmia, and disturbance of nutrition caused by the suppuration and pain from a cancerous tumor, perhaps also the constantly gnawing care with the ever-recurring reflection on the incurable nature of their disease, are factors which may well hasten the course of the malady. Under some circumstances I consider it the duty of the physician to deceive the patient about the incurability of this disease, whether he considers an operation as possible or not; where the physician cannot aid the patient, he should alleviate his sufferings, mental as well as physical. Few persons have the quiet of mind, resignation, firmness, or whatever you choose to call it, to enjoy what remains of life, if they know they have an incurable disease. Although perhaps externally quiet, patients will thank you little for confirming what they may have feared. On this point you will have many trials, and I must leave you in each case to do whatever is dictated by your personal shrewdness, knowledge of men, and your feelings.—Although we may not get rid of the diathesis by the operation, as when, having removed a diseased portion of breast, we fail to prevent new nodules forming in the remaining portion which was previously healthy, or in the other healthy breast (regional recurrence), soon after the cicatrix has healed, still by the early removal of the primary tumor we may prevent the neighboring glands, or the adjacent portion of mamma, from becoming diseased. Few as are the complete recoveries from cancer of the breast after operation, I believe they will grow more frequent when the family-doctor, to whom they are generally first shown, urges operation earlier, for at present they usually let the best time for operation slip by, and the women do not consult professed surgeons, till the local disease and the affection of the axillary glands are so far advanced that a complete operation is no longer practicable. The favorable results from early extirpation of true cancer of the lip should embolden us to remove other cancerous tumors early. If it has hitherto rarely been possible to operate on cancers early and completely, there are still important local causes which indicate even late operations, to prevent as long as possible the advance of the tumor to parts where the disease would necessarily destroy life. Although in most cases there will be local recurrence, this will not take place for months, perhaps for a year, meantime, life will not be directly endangered;

occasionally also it is a question of saving from entire destruction certain parts of the face, as the lips, eyelids, or nose, which may subsequently be replaced by a plastic operation. It would be very unjust to consider such operations useless, because they cannot cure the disease, for they render the patient's life easier and more agreable—if only for a time, still, possibly, for the greater part of the time that he yet has to live. We might be very glad, if, by an operation or other treatment, we could temporarily restore to the pleasures of life a patient with advanced tuberculosis of the lungs, as is the case in operating for some cancerous tumors. In short, there are many cases where we do good by the operation; very often I should consider it wrong to refuse to operate.—We see other cases, however, where it is more difficult to decide. In slowly-progressing cancers of the breast, as in connective-tissue cancers, I consider an operation, which is free from danger, as admissible, but not necessary. If an eyelid be destroyed, or the nose partly or entirely lost, an operation is advisable, in the first case to protect the eyeball, in the second to remove the deformity, and the rather so, because in these slowly-progressing flat cancers of the face frequently there is no local recurrence; in such cases only one thing would prevent my operating, viz., great debility or advanced age of the patient; at least then extensive plastic operations are no longer advisable; even the unavoidable loss of blood, and keeping the patient in bed after the operation, may suffice to extinguish the feeble vital spark. Then comes the question about the admissibility of the operation, where the tumor is in a dangerous location, when an operation is necessary that may end fatally, or at least is just as likely to end fatally as to result in cure. Here we have to drop general reflections, and consider the individual cases; the danger seen in an operation varies greatly with the experience of the surgeon, and the individuality of the patient; one principle we should adhere to: only to operate when after careful examination we can hope to remove all of the diseased part; a half-operation, leaving behind portions of the tumor, should never be done. We should be careful to operate only in healthy tissue, if possible a centimetre or more from the perceptible infiltration, for in this way alone can we be certain of removing all of the diseased part. Occasionally in desperate cases we may prolong life by a bold operation, even if the cancerous tumor be already very large, but generally in such operations we shall see more patients die than will recover.

We have now to criticise the caustics chiefly used in cancers. In the course of time opinions about caustics have differed greatly; at times they were greatly preferred to the knife, again they were entirely thrown aside. The views of most surgeons of the present day as well as my own, incline to the latter view. I decidedly prefer the operation with the knife or scissors, because I then know exactly what I remove and I can judge more certainly if all the diseased part has been excised. Hence, I regard the operative removal of cancer as well as of other tumors to be preferable as a rule. But where there is a rule there are exceptions. In very old, anæmic, or timid patients, caustics may be employed, and, if the treatment be continued till all the diseased portion is destroyed, the result will be favorable. Physiologically caustics would have some advantages; for it is supposable that the cauterizing fluid may enter the finest lymphatic vessels, and thus more certainly destroy the local disease. But this does not occur readily, because the tissue with which the caustic comes in contact instantly combines with it, and its further flow is thus prevented. Formerly it was asserted that recurrence did not take place so soon after the use of caustic as after operation with the knife, but this has not been confirmed; hence I only maintain the above exceptions.

For a caustic I prefer chloride of zinc to all others for destroying cancers; you may use it as paste or as caustic arrows. If it is a surface you wish to cauterize, to equal parts of powdered chloride of zinc and flour you add enough water to make a paste, which you apply to the surface. If you desire to cauterize more deeply, you mix one part of chloride of zinc with three parts of flour or gum and some water, and let them form a cake and dry; this may readily be cut up into small pointed cylinders half a centimetre or more in thickness; with a lancet you make an opening in the tumor and press the caustic arrow into it; you repeat this operation till the tumor is perforated with arrows at about three quarters of an inch distance from each other. In four or five hours this cauterization is followed by moderate, often by very severe pain, which you may greatly modify by giving a subcutaneous injection of morphine directly after the cauterization; the next day you find the tumor changed to a white slough. This becomes detached after five or six days, earlier in soft tumors, later in hard ones. If the cauterization has extended far enough into the healthy parts, after the detachment of the eschar there is left a good granulating wound, which soon cicatrizes; if the carcinomatous mass again grows, the paste or arrows should be again applied, etc.

These cauterizations are occasionally very painful and uncertain as regards the extension of the caustic, but they occasionally are advantageous. Other celebrated caustics are Vienna paste, arsenic paste, butter of antimony, chloride of gold, etc.; iodide of potash, chromic acid, concentrated solutions of chloride of zinc, fuming nitric acid, sulphuric acid, etc., are less employed.

Now a few words of advice about the local treatment of cancerous ulcers which are not, or at least are no longer, suited for operation. In some of these cases the proliferation of the cancerous mass from the wound is enormous, and it often annoys and debilitates the patient; here we may make partial cauterizations or employ the hot iron; by the palliative destruction of the proliferating mass, we occasionally attain tolerably good results. The chief indication for treatment in these patients is suppuration of the ulcer, which is occasionally horridly fetid, and sometimes the pain. For preventing the disagreeable secretion, the hot iron is a good remedy; the smell may be lessened by compresses wet with chlorine-water or purified acetic acid, creosote, carbolic acid, permanganate of potash, sprinkling with powdered charcoal. The latter readily absorbs gases, as you know from chemistry, and is here an excellent remedy; unfortunately, it dirties the wound, so that we abstain from its frequent use. For the pain of carcinomatous ulcers, narcotics have been applied locally, as by sprinkling on powdered opium; but, when injected subcutaneously or given internally, the narcotics act more certainly; hence at last we always resort to morphine for these poor patients. I particularly enjoin on you patience in caring for and alleviating the sufferings of these unfortunates; it is indeed sad for the physician to be able to do so little good in these cases, but still you must not abandon them.

[From recent statistical tables about mammary cancer, it is evident that life is prolonged by operation; but there is no doubt that the disease may also be entirely cured even when infiltrated lymphatic glands have to be removed, or it has to be repeated two or three times on account of relapses. I have had cases where there was no recurrence three years after; in almost all cases where there is recurrence locally, or in the lymphatic glands, it takes place within a year; if only a small proportion of cases is thus cured, operation is justifiable. In most cases there is recurrence after removal, because it is done too late. Some of these cases are operated, without expectation of cure, but to relieve suffering or escape local danger. In operating, some surgeons always remove the adjacent glands, which are apt to be implicated, whether they are at the time diseased or not. Of late, carcinomata of certain regions are removed by the galvano- or thermo-cautery. These are not strictly cauterizations, as the neoplasia is not cauterized, but the removal is made through healthy tissue. *Langenbeck* recommends the thermo-cautery in removing the tongue, as it is a convenient, bloodless operation, and after the diseased tissue is removed the healthy parts are destroyed for some distance, thus increasing the probabilities of a radical operation.

A most important question theoretically and practically is the relation of the lymphatic glands to the primary cancer; we may find in its vicinity glands scarcely as large as a pea, but having microscopically distinct signs of cancerous degeneration. If the infiltrated glands are larger, we may often feel a hard string from the gland to the tumor. Formerly, this was considered as the thrombosed lymph-vessel; now we know it contains lymph and blood-vessels and connective tissue, all more or less implicated in the disease. The glands themselves at first contain disseminated foci of carcinoma tissue, later they change to a cancerous tumor; the capsule is ruptured, the glands melt together and unite with the parts around; adhesions with large vessels, as the axillary vein, are very important. Macroscopic and microscopic examinations show the type and structure of the lymphatic tumors to be the same as those of the primary disease; this is true of all the carcinomata that induce infection of the lymphatics. This correspondence in structure is important in explaining the mode of infection; we have said there were two explanations given: some pathologists suppose that cellular elements of the primary tumor pass by the blood and lymph circulation to various points, and there grow to secondary tumors; of course, this is supported by the adherents of the germ-layer theory; for development of carcinoma in a lymphatic gland, where there is no true epithelium, could only come from the transfer of cancer-cells. Against this hypothesis, *Virchow's* infection theory has lately received some new support. From our present knowledge, this alone is able to explain the facts. Just as I consider the primary development of cancer-cells from elements of connective tissue of the muscles, from endothelium of the lymph and blood-vessels, etc., proved, I also believe that the secondary disease of the lymphatic glands comes by metamorphosis of the cellular elements of the glands themselves, especially of the lymph-granules, cells of the reticulum, walls of the vessels, etc., and that the tumors are induced by true *infection* by seed from the primary tumor, probably carried there by the current in the lymphatics.

Recently *Gussenbauer* has published something on this subject. He believes the bearers of the seed to be small round, pale, or, in melanoma, pigmented granules, which occur in the protoplasm, nuclei of the lymph-cells, endothelium, walls of the vessels, and also free in the lymph-channels. These he calls "corpuscular elements," and says that from the primary tumor they pass into the glands, from which they enter all sorts of cells; their presence indicates the first stage of gland-infection by a tumor, whether sarcoma or carcinoma. Cells of the gland that have taken up these corpuscular elements at once begin to proliferate and approach the type of cells of the infecting tumor; so nodules form just like the primitive growth. *Gussenbauer* regards these elements as "fertilizing germs, which, coming from the infectious tumor, carry certain qualities to the tissue-cells of the glands, so that they no longer bear the type of the mother-tissue, but that of the infecting tumor."

*Gussenbauer* does not go more accurately into the nature and formation of these "fertilizing germs," nor could he discover differences between the germs of different kinds of tumor. The essential part of his theory is, that the corpuscular elements, coming from the tumor, do not themselves form tumor-cells, but that, by entering the lymphatic glands, they excite these to heteroplastic production, or are truly *infecting*. Passage of tumor-cells from the primary tumor into the lymphatics, even if proved, would be much less important than this infection.

When cancer attacks bone, it is eaten away, but not as by pressure from other tumors; there is a peculiar infiltration with tumor-cells.

In suitable preparations, in cross-sections, the Haversian canals appear filled by a mass of cells around the vessels, probably from the wandering cells; there is also proliferation of the bone-corpuscles; the shape of the bone is preserved even when the chalky salts have been absorbed; in the centre there are heaps of cells in concentric rings, separated by basement substance. Gradually the elements preponderate, the bone disappears, but the anatomical structure remains. According to the epithelial theory of *Thiersch* and *Waldeyer*, these are only reaction appearances induced by the encroaching cancerous infiltration; the cells forming around the vessels in the Haversian canals and from the bone-corpuscles seem to me to be young cancer-cells, and the arrangement of the tumor elements in the structure of bone seems a proof of their being formed by proliferation of previously-formed bone-cells.]

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## BRIEF REMARKS ABOUT THE CLINICAL DIAGNOSIS OF TUMORS.

I cannot take it amiss if you are at first somewhat confused by what I have said to you about tumors; if it will encourage you, I may acknowledge that formerly it was the same with me when I was in your present position. Only long study and practice in the differential diagnosis of tumors, for which there is opportunity in the clinic, render it possible to attain any certainty on this difficult point. The consistence of the tumor and its appearance, its relation to the parts around, its locality, the rapidity of its growth, and the age of the patient, are the points from which we start in judging; sometimes one, sometimes another, of these points gives the decision. Let us take an example: A man about fifty years old comes to you, ruddy and strong for his age; for many years he has had a tumor on the back, which formerly gave him no trouble; it has only been inconvenient since it has reached nearly the size of a child's head. The tumor is elastic, soft but not tense or fluctuating, movable under the skin; the latter is unchanged; there has never been pain in the tumor, nor is any caused by the examination. In this case the diagnosis is very easy: from the location, from its seat in the connective tissue, its slow, painless growth, etc., it can scarcely be any thing but a lipoma, or possibly a soft connective-tissue tumor; but the former is most probable. Let us take another case: A woman with a tumor of the breast comes to you; this tumor is hard, nodular, as large as an apple; over the surface the skin is retracted at spots, and is adherent to the tumor. From time to time there has been piercing pain, the tumor is sensitive to pressure, the axillary glands on that side feel hard. The woman is forty-five years old, well nourished, and looks healthy. Here also the diagnosis is easy; it is a carcinoma: 1. Because the patient is at the age when cancerous tumors of the breast are most frequent, while adenoma and sarcoma usually occur earlier; 2. The consistence might point to fibroma, but this very rarely occurs in the breast, and the swelling of the lymphatic glands speaks against this view, and in favor of carcinoma; 3. Carcinomata are painful, as this case is, while sarcomata and fibromata are not so, usually. We might give further reasons for the diagnosis, but these will suffice. Let us take a third case: A boy ten years old has had for two years a slowly-enlarging, moderately painful swelling of the middle part of the lower jaw; at this point the teeth have fallen out without being diseased; the enlargement of the bone is evenly round, and reaches from the first back tooth of one side to the similar point on the other; below, it is hard as bone, above (in the mouth) it is covered by mucous membrane, is firm and elastic. Can this bony swelling be the result of chronic inflammation, of a caries or necrosis? This is not probable: 1. Because the pain has always been slight; 2. Because there has been no suppuration, which would scarcely fail to occur in an inflammation of the jaw that had lasted two years; 3. Because the swelling is more bounded and regular than it is apt to be in bony deposits in caries and necrosis; 4. Because, at the patient's age, osseous inflammation in the lower jaw is not apt to occur unless from phosphoreous poisoning, which has not occurred here. Hence this is a case of tumor; is it an osteoma? The part projecting into the mouth is too soft for this; we may pass a fine needle into the tumor from above. Is it a chondroma? Consistence, form, mode of growth, and age of the patient, agree with this view, but the locality does not; chondromata in the middle of the lower jaw at this age are very rare. It is a central osteo-sarcoma, probably a giant-celled sarcoma; all the symptoms speak in favor of this idea, and you know that these tumors are frequent in the lower jaw during youth. I say you know—I might better say you will gradually learn; and I can only advise you, whenever you have examined a patient with a tumor at the clinic, to read about it when you go home, and to compare the individual case with the general characteristics of the tumors that I have given you. When you have done this for a time, and in the course on pathological histology, under the instruction of your teacher, have examined many tumors, you will obtain a better idea of them, and will have all their peculiarities painted on your memory.

# CHAPTER XXII.

## *AMPUTATIONS, EXARTICULATIONS, AND RESECTIONS.*

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### LECTURE LI.

Importance and Significance of these Operations.—*Amputations and Exarticulations.*—Indications.—Methods.—After-Treatment.—Prognosis.—Conical Stumps.—Prothesis.—Historical Remarks.—*Resections* of Joints.—History.—Indications.—Methods.—After-Treatment.—Prognosis.

GENTLEMEN: We have often had occasion to speak of amputations and resections; so, before closing these lectures, I will explain to you these important operations, by which we remove limbs or portions of limbs which are so diseased that we cannot restore them to health. These operations, which are often so beneficial, even to saving life, are sometimes regarded as a *testimonium paupertatis* of surgery; for cutting off diseased parts is not a genuine cure, if by cure we mean by our skill to restore to its normal state a part of the body which has been changed by disease. However, if you take this high standard for every thing in our art, the bounds of medical science will become very limited. In the same way, you could say a cataract is not curable; for the cloudy lens is not again made clear, but is removed. Many of the most brilliant cures made by dermatologists, where they have used caustics, must be regarded as proofs of the impotence of our art; and the same is true of a case where you prevent a man from suffocating by removing a tumor from the larynx. In the strict sense of the word, the most brilliant "cures" are made in such diseases as syphilis; by antisyphilitic internal treatment, we often cause extensive and old morbid products to disappear in a few weeks, as if by magic. But such undoubted cures are rare in other diseases; we often have to content ourselves with destroying the diseased part, and thus preventing not only the spread of the disease to neighboring parts, but also its injurious effects on the general system. The smaller and more unimportant for the life of the organism the diseased part is, the quicker we shall decide upon sacrificing it. The larger the part to be removed, the greater not only the danger attending the removal, but the more effect will it have on the subsequent usefulness of the patient. This brings an unscientific social element among indications for amputation, which is frequently very important. Thus, a rich man could live, and to a certain extent enjoy life, even after losing all four extremities; for the physiological uses of the limbs may be supplied by the labor of other persons, and labor may be bought. But for any one dependent on the work of his hands or feet, the loss of a limb, or in some artisans the maiming of a finger, may ruin his prospects in life. How can a postman, bricklayer, or turner get on without sound legs, or a jeweler or shoemaker with only one hand? I have often had to remove a finger which had been drawn into the hollow of the hand by a cicatrix, because it prevented the patient from grasping an axe or spade as his business required him to do. How often I have heard patients say: "So you can't cure my foot? I would sooner die than lose it; for what could I do without it? I am a ruined man; I cannot stand it; you shall never take it off!"

But one does not readily die from chronic diseases of the extremities; the pain, continued for weeks, months, or years, finally wears out the strongest; and then love of life, and becoming accustomed to the thought of being able to earn a livelihood even after losing a limb, finally decides most patients to submit to amputation, though sometimes not till it is too late.

The opposition of severely wounded persons to amputation varies greatly; it depends chiefly on the appearance of the injured part, and on the amount of pain. If the extremity be torn to shreds, and pieces of crushed bone be seen in it, there will be little opposition to amputation; the same is true when there are excessive pain and great ecchymosis, and the fingers and toes are immovable. But if this be not the case; if the severity of the injury be only recognized by the surgeon—for instance, if it be a wound of a joint with fracture of a bone, without much deformity or functional disturbance, if the patient can move his toes and fingers and has no pain—it is often difficult to explain to him the necessity for an operation; it requires confidence in the surgeon as in a superhuman being to induce him to permit amputation. You will often find your surgical notions met by insuperable objections. If, after a few days, the dangerous changes which you may have foretold occur, and the patient begs to be amputated, you may sometimes have to say, "It is too late;" but will you be so cruel as to say, "I told you so"? It is a trying moment for the surgeon. If there is the slightest prospect of a cure, even under such circumstances, he will amputate; the hope of saving under such circumstances a patient who has been "given up" is an evidence of youthful and justifiable pride in surgical power. But when we fail to succeed time after time, and grow weary of trying for the rare successes, we become more resigned, and watch with regret the sinking ship without sending out the life-boat of our surgical skill. Seductive as may be the hope of accomplishing wonders by unusual skill, we must still shun the danger of showing our skill to be impotent. For too many mishaps finally annul in every conscientious surgeon the pleasure and trust in his art.

I hope what has been said will make you think seriously, before any important operation, whether you should operate, and how. You must remember that in any serious operation you require the patient to put his life in your hands, and you owe him your best knowledge and skill.

It is difficult to give general indications for amputations and resections; almost any general rule in surgery might be criticised in special cases; but it will be well to epitomize what I have said on these points during the present lectures, and I will add something about the performance of the operations and the after-treatment of the patient.

### AMPUTATIONS AND EXARTICULATIONS.

In some injuries of the extremities, it is certain from the first that the limb must become gangrenous, or that the consequent suppuration will be so great as to seriously endanger the life of the patient. But if primary amputation is not submitted to, and gangrene is far advanced, amputation will probably not prevent death; and the same is true in advanced phlegmonous inflammation with septicæmia. The only hope of success is in cases where you can amputate in perfectly healthy tissue; for instance, when, in traumatic gangrene which has spread from an injury of the hand or forearm to the elbow-joint, you can amputate high in the arm or at the shoulder-joint. Under analogous conditions, similar operations on the thigh or hip-joint are much less favorable.

If conservative treatment has been successfully tried for a time, and then symptoms of pyæmia appear, amputation may be resorted to with some hope of success in the upper extremities, but rarely so in the lower limbs.

In these so-called secondary amputations, a favorable result is more probable when pyæmic symptoms have not appeared, but from excessive inflammation the skin has suppurated so extensively that we cannot hope for the wound to close; or when the patient has fallen into a marasmic state from slow suppuration of large joints or bones.

Injuries of the hands or feet may also lead to amputation, when of such a nature that, under the most favorable condition, they would induce a useless, constantly ulcerating stump. After evulsions or crushed wounds especially, the bones may protrude and the stump may require a regular amputation. The results of frost-bite must be treated in the same way; but in the lower limbs we should not delay amputating too long when the line of demarkation has been formed; sloughing off of considerable portions of the body too often induces septicæmia, which may be prevented by early amputation in cases of gangrene from frost-bite or burns.

In acute idiopathic inflammations of bones and joints, by early diagnosis and treatment, we are constantly learning to preserve limbs by making proper openings for the pus, and fixing the limbs in good position. Still, cases do occur where the patient can only be saved by well-timed amputation; but the choice of the proper time is difficult, as it is a question whether and how long the patient can bear the suppuration and fever.

In regard to so-called spontaneous gangrene, or, as old surgeons called it, grangrene from internal causes, we must carefully consider each case. If the gangrene be due to arterial embolism, and there be general disease, the limb should be amputated as soon as demarkation occurs. In gangrene after typhus and severe exanthemata, we may wait till the patient has somewhat recovered. In true senile gangrene we rarely amputate. If the gangrene be limited to one or a few toes, they may be left to come off spontaneously. If it extend to the tarsus, it is rarely limited to that part; but should it be, we loosen the protruding bones, and strive, with the least possible injury of the soft parts, to secure enough substance to cover the stump.

The chief chronic diseases which give occasion for amputation are the chronic inflammations of bones and joints. Caries of many bones of the carpus or tarsus, of the knee-joint in non-tuberculous adults, of the hip, shoulder, or elbow joints, rather demand resection if any operation is required; amputation is a secondary question.

Extensive incurable ulcers and incurable or frequently recurring pachydermy of the leg often demand amputation, unless the patient is to be condemned to constant pain and to be permanently bedridden.

Large aneurisms of the femoral artery, especially if likely to rupture, if they cannot be cured, would certainly prove fatal if amputation were not performed.

In tumors of the extremities which are firmly adherent to femur, humerus, or tibia, and grow in between the soft parts, we must amputate. Tumors merely attached to the ulna, radius, or fibula, and not extending into the soft parts, may be successfully removed by partial resection, or even by removal of the bone.

Lastly, amputation may be desirable on account of distortion or malformation of the foot preventing a patient from walking.

Now, regarding the *method*, we may operate through the joint, or saw through the bones. Both ways have their advantages and objections. Amputation through the joint appears to be the most natural and simple, and least injurious. The soft parts may even unite to the cartilage by first intention, or the cartilage may suppurate and be thrown off, in which case the healing is by granulations growing from the bone. The medullary cavity of the bone is not opened, so we escape the possibility of primary infection of the medulla at the time of or shortly after the operation.

The objections to this operation are, that portions of the serous synovial sac remain and have little tendency to primary adhesions, and pus readily collects in them after the wound has united. Moreover, the soft parts required to cover the large articular surfaces are very extensive, so that the wounded surface must be very large. In case of the knee or elbow joint, the length of the flap required is such that we might perhaps make a high amputation of the leg or forearm. The stumps left after exarticulation are unfavorable for the application of artificial limbs; for instance, after exarticulation at the knee, the joint of the artificial leg would have to be lower than the knee on the sound side.

In amputations we have the advantage of being able to choose where we will remove the limb, although, from certain empirical reasons, and for greater convenience in applying artificial limbs, certain places are preferred. We generally require less flap to cover the stump from an amputation than from an exarticulation. Sawing the bone is not a very formidable complication of this operation, although in many cases more or less extensive necrosis of the sawed surface results. If the medulla in its cavity or in the spongy substance be infected by a dirty sponge during the operation, or if the soft parts become so adherent that pus forming in the medulla cannot escape, severe acute osteomyelitis results, which may induce septicæmia and death. In more favorable cases the osteomyelitis is limited, and we have necrosis of the bone in the stump; after six or eight weeks the necrosed portion may be removed as a sequestrum;

a bony envelope has formed around it, which replaces the lost bone. When speaking of complicated fractures, we have already stated that osteophytes may form at the amputated end of the bone. Osteomyelitis of the stump is difficult to recognize at its onset; but you may assume its presence if on the third or fourth day after the operation the patient, who has previously been free from fever, suddenly has a high fever with chills and diarrhœa, while the stump shows no signs of inflammation, or perhaps most of it has healed by first intention. Hence, as the cause of the fever is not inflammation of the soft parts, it must be in the bone, unless there be some exceptional complication. At all events, in such cases you should open the stump and expose the bone, to evacuate any pus that may have formed. Occasionally you may in this way save the patient, but usually it is too late; for, from the uncertainty of the symptoms, we rarely have the courage to lay open the beautifully-healed wound, although it would do no great harm even if we were mistaken in the diagnosis.

In amputations and exarticulations the chief points are:

1. To do the operation with as little loss of blood as possible.
2. To arrest the bleeding completely, so that there may be no secondary hæmorrhage.
3. To cover the end of the bone with soft parts, so that they may unite easily and completely.

In regard to the first two points, I have nothing to add to what I have already said. Before the operation *Esmarch's* bandage should be applied; the amputation may then be done without losing a drop of blood. After the operation I twist the small arteries, and close the larger ones by acupressure or catgut ligature. After exarticulation of the femur or humerus I ligate the femoral and axillary arteries, because I have found it such slow work to apply the needles securely.

The end of the bone must be covered with soft parts, which must heal over it; if this does not occur, and the bone projects, the granulations growing out of it either fail to cicatrize and form an ulcer, or, if they do cicatrize, the cicatrix adherent to the bone has so little vitality that wearing an artifical limb soon makes it sore. This is very unfortunate, as it prevents the patient from using the stump. and condemns him to two crutches and pain in the ulcerated stump for the rest of his days. Hence the bone must be sawed higher up than where the soft parts have been divided. In exarticulations the soft parts must always be divided below the end of the bone. In accordance with these principles, the soft parts may be divided as follows, and properly shaped to cover the stump:

1. In the *circular operation* we make a circular incision around the limb, strongly retract the divided soft parts, and saw through the bone; then, letting go the soft parts, they fall over the end of the bone. To obtain this end most certainly it is well to proceed as follows: First divide the skin entirely around the limb, then dissect it up, leaving as much as possible of the cellular tissue with it, and leaving the muscular fasciæ on the muscles. When the skin has been dissected up from two to four centimetres, turn it back like a cuff, and let an assistant strongly retract it with the other soft parts; then cut through the muscles down to the bone with a circular sweep of the knife, at the line where the skin is turned back; the assistant then retracts all the divided parts as far as possible; then with a third circular incision the deep layer of muscles is divided about two centimetres higher than the second cut divided them, the periosteum is divided, and the bone sawed through. If now the parts be allowed to fall into place, three cut surfaces will appear—through the skin, through the muscles, and through the bone, the last at the bottom of a funnel-shaped wound. Where the limbs are thin, the soft parts should reach about six centimetres below the end of the bone; where they are muscular, this distance should be two or three centimetres more. In amputating the forearm or leg, the last incision must divide the interosseous muscles before the bone is sawed.

It will be best for you to make the circular operation as I have just described it, and accustom yourself to make smooth incisions, and to cut by drawing the knife, not by pressing on it. At the same time, I do not mean to say that the circular operation may not be well done in other ways. Sometimes the following modifications are advisable; they differ partly in the shaping of the stump, partly in the mode of operating:

We may amputate a limb *in one plane* as if done with an axe or guillotine; this may be successfully done on the fingers. In the fingers we prefer exarticulation to amputation; but sometimes fingers are thus cut off by machines, such as circular saws, straw-cutters, etc., and the question arises whether the stump will do well without surgical interference: it will do so, but this is merely on account of the anatomical peculiarities of the fingers, where the skin is adherent to the sheaths of the tendons and to the bone, and does not retract, while the tendons retract in the sheath. The cicatricial contraction is concentric, and draws the skin together to the centre of the divided bone, as we might draw a tobacco-pouch. At most other places in the limbs the skin is so movable on the fasciæ, and the muscles on the bone, that after an amputation in one plane the muscles would retract from the bone, and even the skin would retract. After the stump, where the bone projects like the point of a cone, granulates, the force of cicatricial contraction will draw the skin and muscles forward if the latter have not become so united with the bone or skin as to become immovable. As this circular amputation in one plane always leaves conical stumps, it is only done in the fingers or toes.

*Amputation in two planes* is also of limited use. Here the skin is divided and turned back, then the muscles and bone are divided in the same plane; this leaves the stump covered by the skin only. Where the bone is covered by many muscles, they will retract greatly, carrying the skin back with them, so that the end of the bone will lie in about the same plane with the skin-flap; then, in healing, the skin becomes attached to the cone-like section of the muscles, and we have another conical stump. This method is only admissible at points where the muscles will not naturally retract from the bones, or where they and their fasciæ have become adherent to the bones and to each other from long-continued precedent disease. It may answer in amputations of the leg just above the malleoli or just below the head of the fibula, or at analogous points of the forearm; but the skin-flap must be made long enough to cover the stump readily.

The circular amputation *in three planes* first described, where skin, muscles, and bone are separately divided, may be done in various ways. For your first attempts on the cadaver, I advise your doing it as above described. Instead of the last incision through the deep layer of muscles, you may turn back the periosteum two centimetres from the level of the first incision through the muscles, and then saw the bone; the effect on the form of the stump remains the same, whether the deep part of the funnel is covered by periosteum or muscles. This method may be done somewhat quicker and more elegantly if, instead of the three incisions in different planes, you divide the skin, and then have the assistant strongly retract the parts, while you divide the muscles by thin layers. With some practice you will learn to make the funnel just the depth you desire. But if your assistant retracts the soft parts too energetically, and you divide only thin layers, by the time you get down to the bone you will have gone too high, and will have too much flap. If the assistant retracts too feebly, or if the soft parts are adherent to the bone and do not move freely, while you cut rapidly and deep, you get too little covering and have a conical stump.

Lastly, the funnel has been made by cutting obliquely from without inward to the bone. But these methods are not practical, and I will give no further details.

The circular cut is the normal method for all amputations; it is applicable to any part of a limb, although for exarticulations flaps or oval sections are more practical.

2. *Flap Operations.*—From the soft parts we make one or two flaps with which to cover the sawed bone. If we make one flap, with a base half the circumference of the limb at the point of amputation, on the other side we usually make a circular cut in one or two planes. In flap-amputations, also, it is desirable before sawing the bone to turn back the periosteum about one centimetre, and to saw the bone about two centimetres above the base of the flap, so that as the muscles retract the end of the bone shall not press too much against the inner side of the flap.

I prefer making the flap so that while the patient lies in bed it shall hang over the wound without being held by sutures. The lower part of the flap should be of skin, the upper of skin and muscles. The best way of doing this is first to form the flap by an incision through the skin, then retract the skin and cut down through the muscles to the bone, then by two incisions make a circular cut on the posterior part of the leg. The length of the flap should be about one-third the circumference of the limb at the point of amputation, and its breadth about one-half the circumference, or rather more.

The single flap has the advantage that where the cause for amputation, the injury or ulcer, or the line of demarkation in gangrene, is irregular in outline, we may amputate lower than when we perform a circular operation, so that the stump may be longer and the prognosis better.

I do not think the operation with two flaps has any advantage over the circular method. We may make two lateral flaps, or an anterior and posterior one, provided the amount and form of the soft parts is analogous to the circular amputation. Occasionally infiltration of the skin prevents its being retracted well or turned back; then we may incise it in the direction of the long axis. This would make of the circular an operation with skin-flaps, having the funnel shape within.

Flaps for covering the stump with skin alone are not good, for long flaps of this kind readily become gangrenous at the edge, and, there being no muscular layer between the skin and the sawed end of the bone, the latter readily causes ulceration and perforation of the flap. It is true, this is no great misfortune, as the exposed portion of bone either necroses and separates, or soon granulates and cicatrizes; but in either case the cicatrix becomes adherent to the bone, and the subsequent use of the stump may give rise to tedious ulceration.

The method of forming the flaps by transfixing the limb with a long, pointed knife, and then cutting, usually results with beginners in making a muscular flap, which is occasionally tongue-like, is covered with too little skin, and does not well cover the wound. If before entering the knife we have the skin strongly retracted, and pass the knife flat alongside the bone, we may make good flaps; but it requires more experience and practice than the former methods.

Flap operations are possible at any part of a limb, but are not everywhere advisable. By drainage-tubes we may lead off the secretions, even in flaps formed from below. If the flaps do not unite by first intention, the after-treatment is always tedious; for we have to guard against cicatricial contraction rolling them in.

3. Finally, in a third method, the wound made is between a circle and a flap; it is called the *oval amputation.* The plane of incision of the oval lies obliquely from above down; the upper part of the oval is more pointed, the lower more rounded. After making the incision through the skin, it is to be drawn back, and the soft parts and bone are to be divided, as in the circular operation. For amputations the oval incision is rarely used, as it has no advantage over the circular or flap operations. In exarticulations of the fingers and toes at the metacarpo- and metatarso-phalangeal articulations, or of the big toe or thumb, the oval incision is very useful. In exarticulations at the shoulder or hip joint, I would only employ this method when there was not skin enough to form a flap.

I have still something to add in regard to preparations, assistants, choice of instruments, and after-treatment of amputations.

While the patient is being anæsthetized, or previously (for it is hard to bring some patients under anæsthesia when their attention is excited by manipulation of the affected part), we carefully cleanse the part with soap and water, especially at the point of operation. Then the bandage for preventing hæmorrhage is applied, and taken off again except the upper band. Now one assistant holds the upper part of the limb, another the lower. In amputations the operator stands so that he may assist in retracting the soft parts, and that the part to be amputated falls to his right; in exarticulations he should stand so that he can himself, with his left hand, control the movements of the limb to be removed.

For amputations and exarticulations of the toes, we use small knives with blades four or five centimetres long; they should not be too much curved in front, or the point will not enter the joint readily. For exarticulations of the hand and foot, as well as for amputations of the lower half of the forearm and leg, we choose a knife with a blade about 15 centimetres long; for the upper part of the forearm, the arm, upper part of the leg, and the lower part of the thigh, the blade should be from 15 to 25 centimetres long; for high amputations and exarticulations of the thigh, it should be 25 to 35 centimetres long. If you have two small knives with blades 5 centimetres, and one each of 15, 25, and 35 centimetres, it will be enough. In amputating I do not like changing knives, and so prefer having the cutting edge somewhat rounded in front, so that the skin may be dissected up with the point of the same knife. Other operators prefer doing this with a scalpel, then taking another knife to divide the muscles, and still another for the periosteum. For pushing back the periosteum, I use a raspatorium, though sometimes this may be done by the nail alone. A skilled assistant will with his hands retract the soft parts sufficiently to give the operater room to cut and saw; but pieces of clean linen may be used for this purpose. Some operators take pride in amputating even thick limbs with small knives, thus pushing the simplicity of instruments to the utmost point. These points, while not unessential, depend greatly on habit and tradition, and each one may follow his own taste.

Saws for amputation are usually bow-shaped. The bow should not be too high, or it will make the saw unsteady; the handle should be broad and lie securely in the hand. The blade should not be more than two centimetres broad, and the teeth should be bent outward, else the saw will catch; and it will be still more apt to do so if the assistant holding the lower part of the limb raise it instead of depressing it somewhat. After sawing the bone, I usually cut off the sharp edges with bone-nippers.

When the amputation is completed, the vessels are twisted, compressed with needles, or ligated; the instruments required for this should be all ready. In one of these ways we first close all the vessels we can find, then relax the elastic band or tourniquet, doing it in such a way that the assistant can renew the compression if the bleeding becomes excessive; then we apply acupressure or ligatures to any other arteries we see bleeding. In amputations of the thigh or arm we may have venous hæmorrhages, as the valves are insufficient. The veins may be ligated or compressed by needles; torsion of veins is dangerous. Arterial hæmorrhage from the medullary cavity of bones is very unpleasant; it is rarely severe. But either poking into the medulla with forceps or firm pressure with a sponge is dangerous; and we should entirely avoid the application of styptics, especially liq. ferri. I advise letting the bleeding alone till all other vessels are cared for; if by that time it has not ceased spontaneously, we may compress the main artery of the limb for a while with the finger. During the dressing we should only use new, soft sponges.

We should wait till the bleeding is entirely arrested; it is even desirable to leave the wound exposed to the air for a time. After circular or oval amputations, the wound is generally united in a vertical direction. I apply from two to four sutures in the upper part of the wound, leaving the lower part open. I secure flaps in the position they are to occupy by from two to four sutures, previously placing a drainage-tube dipped in glycerine in the wound across the bone, so that the two ends project from the angles of the wound.

[In amputations at the upper third of the leg, the spine of the tibia is apt to press on the soft parts; to avoid this, the tibia may be sawed obliquely downward and backward: this is easier than cutting it with forceps.

Instead of applying *Esmarch's* bandage, by which pus may be forced into the veins, or blood-clots there be driven into the circulation, we may have the limb held vertically for ten or fifteen minutes, and then tightly apply an elastic bandage above the proposed point of operation.

If skilled in finding the vessels, you may ligate them, apply drainage-tubes, sew up the wound, and apply an exact compression dressing, while the limb is kept elevated, and then remove the elastic bandage; but do not try this with your first operations, as it requires practice. It is better, after removing the bandage, to ligate any small vessels that have been overlooked, wash off the wound with a four-per-cent. solution of carbolic acid, and then unite it exactly. The ligatures of disinfected catgut or silk are cut short, the edges of the skin are brought together so that the line of union may be vertical; after from two to four drainage-tubes have been inserted at proper places, so as to remove secretion entirely and by the shortest way, these are cut off level with the skin and fastened there, first satisfying yourself that they act well by passing a two-per-cent. solution of carbolic acid through them. Then *Lister's* dressing is to be applied, or the iodoform dressing of which we have already spoken; the parts where the drainage-tubes are to lie, and the end of the bone, should have very little iodoform applied; then the edges of the skin are to be united and more iodoform applied, with greased cotton and gutta-percha paper outside.

The stump should be kept elevated for a day, then laid nearly horizontal. The dressing is usually changed in twenty-four hours, then, if all goes right, not for four or five days, when the drainage-tubes should be removed. After this the dressing is only to be changed when permeated by secretion, or from some accident.

Normally the stump should be entirely free from pain, redness, and swelling, and there should be no febrile reaction, except a slight rise the first and second day. The sutures are generally removed at the third dressing; after a fortnight, even if the wound is not entirely healed, we may apply a simple compress wet with solution of acetate of alumina, or some adhesive strips. The iodoform dressing is not usually changed for four or five days, and the drainage-tubes are then taken out, unless absorbable tubes of decalcified bone have been used, then the dressing may be left longer.]

Should the stump swell, or the patient become feverish, the adhesions of the wound should be broken up with the finger, and any pus that has collected be allowed to escape. If there are neuralgic pains or frequent twitchings, subcutaneous injections of morphine may be made.

If arterial *secondary hæmorrhage* occurs within twenty-four hours, the artery should be sought for and closed; if it comes later, in the second or third week, when the wound is granulating, it is best first to seek the bleeding point and close it firmly; if this attempt is unsuccessful, and the hæmorrhage recurs after prolonged digital compression, the main artery of the limb should be ligated.

Treating the stump in the water-bath was attended by so many difficulties that it was soon given up.

When treating of circular amputations made in one plane, we spoke of the unfortunate occurrence of *conical stumps*. They may be due to unsuitable incisions through the soft parts or deficiency of soft parts; but these are not the only or even most frequent causes. In marasmic patients there is sometimes such an atrophy of the soft parts of the stump that they grow thinner and shorter, and sink more and more on the bone.

If we see that a conical stump is forming, we may apply adhesive plaster and weights, as in coxitis, to draw the skin down, or at least to aid the concentric contraction of the granulating surface by freeing it from the opposition. If the patient bears this without pain in the stump or fever, it may be of benefit; but if these symptoms appear, it must be abandoned. If, as a result of osteomyelitis, extensive necrosis of the stump occurs, it is indeed shortened, but the osteophytes which have formed prevent too much shrinkage, and they do not atrophy for years. My experience does not show that the stump becomes less conical by the detachment of the sequestrum; usually an operation is required. I split the mass of granulations upward into the skin and downward to the bone, then push the raspatorium along the bone, separating the periosteum and osteophytes from the bone so far into the soft parts that they may cover the stump of bone which shall be left. In sawing, I use a chain-saw, whose ends I bring out above while the loop is around the bone. In limbs with two bones, this subperiosteal resection or amputation is done on both bones. Care must be taken that the secretion from the periosteal canal from which the bone is removed has a free escape ; it is much inclined to close in front by first intention ; then pus may collect deeper in, decompose, and cause osteomyelitis. I had such an unfortunate case in the army-hospital at Mannheim, in a soldier who was amputated successfully for a severe injury of the knee, and finally died in this way. At that time I did not know of this danger from subperiosteal resection of an amputation-stump, all the previous cases that I had operated on in this way having done well.

Observation of old amputation-stumps shows that they change considerably in the course of years : some grow very thin ; the muscular covering or flaps atrophy from disuse, so that only the skin remains. In the course of years most stumps become conical even if covered with skin alone. This is the more certain to occur, the more poorly nourished and marasmic the patient, and especially in those who have been amputated for caries of the joint, and who subsequently have caries of other bones or in the stump, or pulmonary tuberculosis or lardaceous disease. The bones of such stumps atrophy, and their cortical layer becomes thin. Short thigh-stumps are about the only exception. If these are used much in walking, the muscles going from the pelvis to the thigh develop, the skin and cellular tissue participate in the good nutrition, and the stumps become larger than they were shortly after the operation. From most old amputation-stumps being covered only by skin, while the muscles have disappeared, some have asserted that it is entirely useless to employ muscle for covering the stump ; but we have already shown that they would not heal so well.

In Lecture IX. we treated of neuromata in the amputation-stump.

Regarding the *prognosis* for amputations, we can only say in general terms that they are the more dangerous the nearer they are to the trunk. Much depends on the general condition of the patient at the time of operation. Amputations for injuries are always less successful than those made for chronic diseases ; but in each case there are many points that we will not lose our time about here.

Surgeons pay too little attention to the subsequent fate and the artificial limbs of those they amputate. You will hear many complaints from these patients. Pains in the stump with each change in the weather, excoriations of the cicatrix, pressure of the artificial limb at one place or another, and constant repairs of it, are the most frequent complaints. Some suffer for years from the sensation of still having their limb; for instance, after amputation of the thigh they exclaim: "I have a pain in the little toe; the big toe is being torn; my foot lies in a bad position," etc. During the first days and weeks after amputation these sensations are the rule, and are so decided and strong that by covering the stump we may deceive patients for weeks about the loss of their limbs; but I have seen patients who had the same sensations after years.

As regards the substitute for the limb, much depends on the patient's position in life and his means, not only for buying a limb, but for keeping it in repair and replacing it when worn out.

Artificial arms and well-imitated hands are articles of adornment and luxury. Active movements of the fingers have not been attained, but mechanism for grasping has been arranged with springs which are opened by the other hand. I will not enter into more detail. For the arm or forearm, a workman may have a leather case held in place by straps, and having a solid piece of wood at its lower end, into which may be fastened hooks, etc. On Sunday he may put on a hand carved from wood. It is astonishing what intelligent persons can accomplish with such apparatuses. I have a long, beautifully-written letter from a man for whom I amputated both hands. He was an engineer, and got his hands caught and crushed in a rapidly-revolving water-wheel. Subsequently, without hands, he earned his living by writing.

As regards the lower limbs, there are few stumps on which the patient can bear the entire weight of the body; and these are the stumps after amputations and exarticulations of the foot, and sometimes after exarticulations at the knee. In all other cases the patients rest on the condyles of the tibia or tuber ischii, which bony parts are supported on a firm, cushioned ring, which forms the upper end of the shield of the artificial limb, and into which the stump is introduced. After amputations of the leg, it is desirable to divide the weight of the body on these two points. Another way is for a patient whose leg has been amputated to rest the bent knee on a wooden leg; this, of course, prevents any motion at the knee-joint. In regard to the construction of artificial limbs and wooden legs, I will say nothing, except to add that for their use a certain amount of skill and intelligence is required, as well as pecuniary means for attending to the repairs so often required by any artificial limb. Hence, for working-people, as most of our hospital patients are, it is better to have firm wooden legs. Even many from the higher classes, who have been amputated, and have worried over artificial limbs for years, finally resort to wooden legs. Walking with an artificial leg and a wooden one differs so much, that one who has been accustomed to the latter for years can only use the former after very patient trial.

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Simple as the operations for amputation and exarticulation now seem, we must remember that from Hippocrates to the present time progress has constantly been made in them. That large portions of limbs could be lost without danger to life was first taught by their spontaneous detachment by gangrene; the first amputations were made for the purpose of removing such gangrenous limbs, and the bone was sawed at the line of demarkation. The indications for amputation grew very slowly. What especially retarded the introduction of this operation was not knowing how to check the hæmorrhage with certainty; styptics and the hot iron answered for the leg and forearm, but not elsewhere. Hence the progress of amputations depended on that of the methods for arresting hæmorrhage. The greater amputations were only ventured on after the introduction of the ligature and tourniquet. The method of amputating limbs by strangulating them with a ligature was first introduced by *Guy de Chauliac* and improved by *Ploucquet*. Of late this method has been tried again by the *écraseur* (*Chassaignac*), the galvano-caustic (*Von Bruns*), and the elastic ligature (*Dittel*), but has met with little popularity. Later surgeons particularly directed their attention to amputating as rapidly as possible, so as to cause the least pain, and to dividing the soft parts so as to avoid conical stumps. Now that we have anæsthetics, and can avoid hæmorrhage by the elastic bandage, rapidity in amputations and exarticulations is a matter of small moment. Attention is turned to the formation of the stump, and since the beginning of this century to attempts to secure healing by first intention, and especially to the avoidance of any infection from without or from the secretions of the wound, and to escaping pyæmia, the most dangerous enemy of amputations. The latter points now chiefly claim our attention, and recent proposed changes in the method of operating all have them in view.

The first method which was used in *Celsus's* time was a circular incision with retraction of the skin. This was gradually improved on. *Lowdham* (1679) is usually regarded as the originator of the single flap, which method was perfected by *Verduin* (1696). *Ravaton* and *Vermale* are said to have been the first to use two flaps.

The oval amputation was first made by *Scoutetten*. You will find very accurate accounts of amputations in *Sprengel's* histories of operations, and in *Linhart's* excellent work on operations, which I cannot too highly recommend.

## RESECTIONS.

I will now make a few general remarks on resections. As previously stated, sawing, chiseling, or gouging out diseased or injured pieces of bone from the body of the bones, is called "resection in the continuity." Most operations of this nature were mentioned when treating of complicated fractures, necrosis, or caries; as were the so-called osteotomies for orthopedic purposes. You will see these operations so often in the clinic that I will not describe them here; they are mostly simple. The indications for them appear from what has been said.

We have also spoken of "resections of joints." I have already told you that these operations, which in civil practice occur especially for caries, have different results and indications for each joint. The same is true of resections of joints in gunshot-wounds; each joint has its special resection history. Resections, especially resections of whole joints, are of much more recent date than amputations. The first excision of a carious head of a humerus was made by *White*, 1768; resection of the elbow-joint by *Moreau*, 1782; of the head of the femur by *White*, 1769; of the knee-joint by *Park*, 1762. But at first these operations were not popular; they were said to be too difficult, tedious, and painful, and it was thought that the final results would not be good. It is only within the past thirty years that they have been accepted by surgeons, and the methods of their performance are still being improved. At first it was merely attempted to remove the affected portion of bone without loss of the limb, so that the parts might heal. Later, attempts were made to retain the function of the false joint left after the resection, by judicious selection of the lines of incision, method of operating, and after-treatment. Surgeons even went so far as to excise stiff joints that were all healed, so as to substitute for them movable false joints. Possibly, for a time, we held too hopeful views of what was attainable by these operations; but wonderful cures have been accomplished, and with the increased attention now given to them we may expect the indications, mode of operating, prognosis, and after-treatment to be more defined.

In resections, the incisions should be so directed that no large vessels or nerves, and as few muscles as possible, may be injured, and still space made for freeing the joint and sawing the bone.

When these operations were first done, they seemed so difficult that it was thought the joint should be exposed by large, deep flaps, so that ligaments and muscular insertions could be readily divided, and the ends of the bones sawed off. Later, as more was thought of making a useful joint, the operation was more carefully made; oblique sections of tendons and large wounds were avoided; the periosteum was preserved as much as possible, and also its connection with the muscular insertions, by using the raspatorium instead of the knife; and in chronic inflammations operations were done in the thickened tissue, where they were followed by less inflammation and febrile reaction than in healthy parts. *B. von Langenbeck*, more than any one, developed the indications for resections of joints, and perfected the methods of performing them; he also introduced the simple longitudinal incision, which is now generally used for resection of the shoulder, elbow, and hip; for the knee an anterior flap is made, with a broad base above.

The instruments used for resections, except the chain-saw (of *Jeffray*), are the same as recommended by *Von Langenbeck:* a strong knife, five to seven centimetres long, with straight edge and thick back; this knife is introduced to the bone, and the entire incision made at one stroke; with a broad, small, more or less curved, half-sharp raspatorium, the periosteum is scraped from the bone; the articular ligaments and some muscular attachments cannot always be detached in this way, and have to be divided close to the bone. When the ends of the bone have been entirely denuded of soft parts, they are sawed off, being held by bone-forceps, while the soft parts are held back with blunt hooks. Sharp edges of bone are to be removed with bone-nippers.

Before the operation the patient is anæsthetized, and the limb to be operated on is rendered bloodless by the elastic bandage and carefully cleansed. After the operation the hæmorrhage is to be carefully checked, and the wound washed with new, clean sponges; then the limb is placed in an apparatus to keep it perfectly immovable, but not too tight, allowing the wound to be free and the secretion to escape readily; the patient's position should be easy, and changed without disturbing the dressing.

I have not found it well to fill the wound with charpie and apply a bandage before loosening the pressure above the wound; for in so doing the whole bandage is so filled with blood that it must be soon renewed. I prefer first checking the bleeding completely by ligation, acupressure, ice-water, etc.; then I introduce drainage-tubes, dipped in glycerine or carbolic-acid solution, to conduct the secretion into vessels placed beneath.

When possible, I apply a plaster-of-Paris bandage before the operation, make openings of the proper size at the point of operation, then slit the bandage down one side when it is dry and remove it for the operation, and have a plaster shell to put on after the operation, in which the limb can be suspended or laid on a *Ris's* supporting splint. Other operators prefer prepared wooden or iron splints; the same end may be attained with the most varied materials. After resection of the hip-joint no bandage is usually required, extension by weights answering the purpose. In some cases I have found *Lister's* dressing very useful.

Resection-wounds are always rather complicated excavated wounds; they always heal by granulation and suppuration after a long time. This is bad for resections in feeble, marasmic patients; moreover, in such patients we are never certain that caries will not attack the adjacent bones or the sawed surface, or that the wound will not ulcerate.

The shortest time for healing after resection will probably be two or three months. Indolent fistulæ often remain for months or years.

Recently great attention has been paid to the final result of resections of joints. The resulting false joints may become so relaxed that they cannot be actively moved, and the limb hangs useless; other loose joints are somewhat movable; then there are joints which have nearly normal mobility and power; and lastly, anchylosed joints, which are nevertheless more useful than the first mentioned.

The final result depends greatly on the amount of bone removed, the amount of regeneration of bone, the care observed in dividing the muscular insertions, and the muscular power of the patient; it is also greatly influenced by gymnastic exercises, electricity, baths, and application of suitable apparatus. But as these vary with the different joints, and different methods and apparatus are required, these matters can only be explained when treating of resection of individual joints.

Prognosis as regards life is the same as in amputations. Resections on account of caries usually run a more favorable course than those for injuries. The danger increases with the proximity to the trunk. [But here, even more than in amputations, has Listerism proved beneficial. Under this, and the treatment by iodoform, the period required for healing is very much lessened.]

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